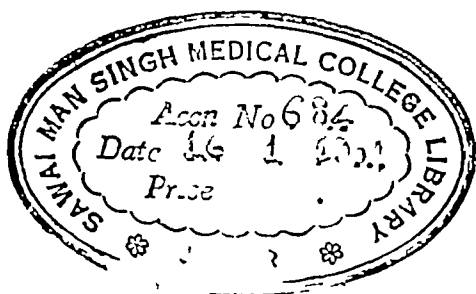


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THE WASTE OF SLAUGHTER AND THE RAGE OF FIGHT

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Inhuman discord is thy dire delight,
The waste of slaughter and the rage of fight
Jove to Mars—

Homer's Iliad, Book IV

IT SEEMS particularly fitting at this time to talk about the military problems of thoracic surgery. This Association was founded at the close of World War I, only a few years ago we concluded a second major war, World War II, and are now enmeshed in another whose casualties are already over 106,000. Inasmuch as we shall probably continue in a military way of life for some years hence, I believe, therefore, we should assume certain responsibilities in the preparation of young men for their future tasks in the Armed Forces.

Military surgery is as old as man himself. At the siege of Troy the Greeks had two surgeons in their army and when one, Machaon, forsook his primary calling and was wounded while joining his fellows in arms to repel a particularly fierce assault by the Trojans, great consternation fell upon the Greeks lest they lose him.

A wise physician skilled our wound to heal,
Is more than armies to the public weal.

Idomeneus to Nestor—

Homer's Iliad, Book XI

In his descriptions of the numerous battles in that matchless epic, Homer depicts the battle wounds and deaths with exquisite vividness and anatomic precision, and there is great drama, pathos, and exultation as he describes the mortal wound.

Deep in his breast above the pap it went,
Amid the lungs was fixed the winged wood,
And quivering in his heaving bosom stood

Thaos—

Homer's Iliad, Book IV

From the Department of Surgery, Columbia University
Presidential Address, Thirty-second Annual Meeting of The American Association for
Thoracic Surgery, Dallas, Texas, May 8-10, 1952.

His lab'ring heart heaves with so strong a bound,
The long lance shakes, and vibrates in the wound,
Fast flowing from its source as prone he lay,
Life's purple tide impetuous gushed away

Idomeneus slays the young Alcathous—
Homer's *Iliad*, Book XIII

Then drew the weapon from the panting heart,
Its reeking fibers clinging to the dart

Patroclus slays Sarpedon—
Homer's *Iliad*, Book XVI

The age of hand-to-hand combat persisted for still another twenty-five hundred years. The only weapons to be added were the catapult and the crossbow.

In A.D. 1215, however, a new extremely potent and destructive agent appeared destined to change the entire character of arms and of war itself. Although first introduced by Genghis Khan against the Chinese, gunpowder was first heard of in the West at the battles of Cressy (1246) and Agincourt (1415), where cannon and gunshot were employed.

The gunshot wound was first described by Brunschwig in 1497 and again a few years later by Vigo as a poisoned or envenomed wound. This idea was finally dispelled by Peré, in 1536 when he observed that gunshot wounds dressed with simple emollients ran a far more kindly course than those treated by the standard method of cauterization with boiling oil. The surgery of gunshot wounds was stated with amazing clarity and vision by an Elizabethan surgeon, Thomas Gale, in 1586 and here is the very essence of debridement as we know it today. After proving that they were not poisonous per se, he says:

And there are two intentions properly hereto belonging. The one is to deliver the wound of all such things as are not agreeing to nature. The other is restoring of such things as are lost or perished. I call things not agreeing with nature not only shot, iron splinters, or shivers of wood, cloth, dust, oil, or such like, but also the clots of blood, matter, bruised flesh, and such like as have no society with nature. The restoring of that which is lost, is properly the office of nature as to engender flesh, blood, and such like parts as are to be engendered. Notwithstanding the surgeon herein is Nature's minister and shall labor to bring the part to his right temperature and it so reduced to conserve it in the same estate.

Again it was more than two centuries later that the next great advance in military surgery was made by an extraordinarily brilliant young French surgeon, Baron Dominique Larrey. Not only was Larrey a very great surgeon but he was also a most able administrator and a military genius. He was a zealous advocate of prompt surgery for the seriously wounded and strongly condemned the prevailing custom of leaving the wounded on the field until after the battle. Therefore in 1795 during the Italian campaign he introduced the flying ambulance. This innovation consisted of two- and four-wheeled ambulances with companies of men trained as litter bearers and in simple first aid measures. They carried the wounded to forward dressing stations or ambulances where the seriously wounded were operated upon and the lesser casualties evacuated to the rear. We thus see the germination of the three-point system of forward surgery: triage, early surgery for the non-

transportable wounded, and near-by real installations for the transportables, which was responsible in great measure for the very considerable decrease in mortality in chest and thoraco-abdominal wounds in World War II. Larrey also noted that penetrating wounds of the chest with an opening larger than the glottis did badly. He therefore advised that these patients be placed on the wounded side to permit the blood to drain out and that the chest be closed and described several such cases with dramatic improvement and recovery following this treatment. This was a truly notable advance in the surgery of penetrating wounds of the thorax, and the first recognition of the dangers of an open chest.³

Although an excellent English translation was available in 1814, the United States Army seems to have profited little from the observations of this remarkable surgeon. At the outbreak of the American Civil War there were two two-wheeled ambulances assigned to each regiment but they were at the complete disposition of the Quartermaster Department, the result was that the Medical Department rarely, if ever, was able to use them. On the recommendation of the Sanitary Commission, William A. Hammond was appointed Surgeon General in 1862. He was a man of considerable vision and of great professional and executive ability, and promptly nominated Jonathan Letterman as his Inspector General. When this appointment was not confirmed by the Senate he named him Medical Director of the Army of the Potomac. Together they accomplished a revision of the whole evacuation system based on the experience of Larrey, so that at Antietam all the wounded were collected within twenty-four hours. Furthermore the type of sick and wounded reports instituted by them were later of tremendous value in compiling the Medical and Surgical History of the War of the Rebellion.⁴

In this war there were 253,142 wounds of which 20,607 (8.1 per cent) were in the chest.⁵ This ratio of chest wounds to the total number has remained approximately the same in all the major wars during the past one hundred years with the exception of World War I, when it dropped to 3.8 per cent for Great Britain and 2.5 to 5 per cent for the United States. This is quite inexplicable in view of the German, Russian and French figures of 6 to 9 per cent. Again, in World War II our incidence was about 8 per cent.

It is difficult for us, ninety years later, to picture the status of surgery in the war between the States. Anesthesia was limited, antisepsis and asepsis undreamed of, our medical schools poorly organized and administered, there was scant dissemination and exchange of medical knowledge, there was no intern and residency training and no order and system at all within the Army. The over-all mortality for 20,607 chest wounds was 27.8 per cent, and for 8,404 penetrating wounds 62.5 per cent, chiefly from hemorrhage and empyema. As to thoracentesis: "This operation was occasionally resorted to during the war to relieve the effects of effusion resulting from acute and chronic pleurisy, and more frequently on account of effusions consequent on traumatic pleuropneumonia, or the lodgment of foreign bodies within the chest." One hundred twenty wounds involving the diaphragm are recorded, recovery was rare

Of the major vessels, there were twenty-five ligations of the subclavian artery with five recoveries, and thirteen ligations of the axillary artery with thirteen fatalities. All died of secondary hemorrhage and sepsis, and in only two instances were *both* ends of the artery tied.⁵

As we proceed, we note that in 1892 a change in the type of arms occurred, the high powered rifle replaced the old muzzle loader, and about the same time the jacketed bullet appeared with the thin steel, nickel, or copper jacket. The surgeon was the important figure in the Civil War, but after this he began to fade into insignificance with the rise of sanitation and preventive medicine. The vital necessity for these was emphasized during the Spanish and Boer wars, when typhoid was rampant and sanitation almost nil. Thus we find ourselves at the threshold of the twentieth century with modern surgery in its early formative years, and sanitation and preventive medicine just awakening. As we entered World War I, the treatment of wounds was not very different from that of the Franco German War of 1870—dress the wound, drain, or occlude the part. We were completely unprepared for the extravagant use of artillery, high explosives, and high-velocity missiles that were soon manifest after the outbreak of war in 1914. Until that time the preponderance of wounds had been caused by bullets and low-velocity missiles. Now the pendulum swung the other way and 70 per cent of the wounds were caused by high explosives and high-velocity missiles.

Before the United States entered the war, Pierre Duval of the French Army made some important observations.⁶ At the outbreak of the war it was taught that thoracic wounds should not be operated upon until after the patient had reacted from his initial shock. Duval, however, realized the dire results of this policy, and stated that the chief causes of death at the Aid Posts were hemorrhage and mechanical asphyxia due to an open thorax, he further noted that the septic sequelae of shell wounds were far greater than in those from bullets, which were fatal either immediately or else ran a relatively benign course. The mortality from the former was approximately 30 per cent, from bullet wounds somewhere between 5 and 15 per cent. In 3,453 hospital admissions in the Army areas the over-all death rate of admissions to field surgical units was 20 per cent, while the average mortality of chest wounds in the forward units of the field hospitals was 45 per cent. He realized that it was difficult to state clearly the indications for immediate operation, but urged that if signs of hemorrhage persisted, the wound should be promptly opened and the damage sought for and repaired, whether in the chest wall or lung. As debridement for wounds of the soft parts and extremities was already being practiced with considerable success, he stated that the lung and pleura were contaminated just like other wounds, and therefore the treatment should be the same. In one series of 500 cases of hemothorax, 195 were infected and in a personal series of 193 of his own in the Battle of the Somme there were 49 bullet wounds with no infections and 144 shell wounds with 34 infections. He advocated further that all shell wounds and retained foreign bodies be promptly operated upon under local novocaine block and that the chest be closed tightly without drainage. "As technique improves," he said "the

general surgery of gunshot wounds is being applied little by little to wounds of the lungs. Let us dare to hope that those who follow in our steps may soon have many more numerous and more brilliant successes."

As we entered the war we had Duval's work upon which to build, plus the general experience of both the Allies and the Germans. In 1917 a Special Services Branch of the Medical Department was established with eight divisions. In the American Expeditionary Force, General Orders 88, General Headquarters, 1918, was the first order setting forth this organization with a Director of Professional Services appointed and the positions of Chief Consultant of Medicine and Surgery specified. Partly because of the times themselves, and partly because of the epidemic of influenza with pneumonia and the existing epidemics in the Camps in the United States of measles and pneumonia, attention was focused chiefly on the treatment of infection, and this was particularly so as regards the chest, and, as we know, empyema became such a problem that the Empyema Commission was created.

There were only 4,595, or 2.5 to 5.6 per cent, of chest wounds listed of the total of 174,296 wounds.⁸ Nevertheless, the mortality in this group was 24.06 per cent, and of the penetrating chest wounds alone, 47.7 per cent in the American Army (Tables I⁹, II¹⁰, and III¹⁰). All emphasis was placed upon the prevention and treatment of infection, particularly the latter. With the conditions as they were, and the hemolytic streptococcus and respiratory infections so prevalent, this was inevitable. Still, as we look back upon that era, it is somewhat perplexing that more attention was not directed toward the physiology and the spatial dynamics of the chest. Anesthesia, we realize, was still limited and blood transfusions, with the concept of blood banks, were just coming into being through the work of Captain O. H. Robertson,⁸ but it was not until World War II that we were to appreciate the full value of both of these extremely important agents. At that time the indications for immediate operation of thoracic wounds were (1) aspirating wounds, (2) large retained foreign bodies, (3) severe injuries of the bone, (4) complicated lesions of the diaphragm, (5) extensive hemorrhage and suspected infection—particularly anaerobic. A firm and airtight parietal closure was imperative.

TABLE I BATTLE CASUALTIES WORLD WAR I

	NUMBER	PER CENT	DIED	PER CENT
Abdomen and pelvis	3,546	2.0	1,536	43.3
Thorax	4,595	2.6	1,105	24.06
Face	3,607	5.0	316	8.6
Genitals	798		69	8.6
Head	10,453	6.0	1,456	13.9
Neck	594		65	10.9
Spine	598		334	55.8
Back	2,553		308	12.0
Upper extremities	62,238	36.0	2,412	3.9
Lower extremities	78,032	44.7	5,722	7.3
Unspecified	2,282		146	6.4
Total	174,296			

Carter B. N. and DeBakey M. E. J. THORACIC SURG 13: 271 1944 Current Observations on War Wounds of the Chest

TABLE II WORLD WAR I HOCHÉ REVIEW OF ELEVEN MILLION WOUNDS IN ARMIES OF GREAT BRITAIN, FRANCE, UNITED STATES, AND GERMANY

	NUMBER	PER CENT
Thorax	660,000	6.0
Mortality		56.0
Berry F B Treatment of Injuries to the Chest Am J Surg	54	280 1941

TABLE III WORLD WAR I HOCHÉ WOUND DISTRIBUTION IN 12,350 KILLED IN ACTION

	PER CENT
Head	47.0
Chest	20.0
Abdomen	11.8
Berry F B Treatment of Injuries to the Chest Am J Surg	54 280 1941

Toward the end of the war in discussion in the Allied Medical meetings in Paris permanent airtight closure was considered imperative with control of pleural effusions by closed drainage so as to permit prompt pulmonary expansion. There was also a growing opinion in favor of open operation in the treatment of extensive hemothorax, particularly if massive clotting had occurred, though aspiration was the initial treatment when possible, as it was recognized that it led to early recovery and return to duty. The bad results of organized hemothorax were noted, and also the fact that about 25 per cent of the hemothoraces became infected, hence thoracotomy with mechanical pleural cleansing and tight closure was recommended. This might be done initially according to three methods: first, by mere débridement of the wounds of entrance and exit, second, by a formal thoracotomy at the site of election with repair of the wounds of entrance and exit, and third, with removal of foreign bodies and resection and suture of lung when indicated. At this time postoperative airtight drainage was advised as better than repeated aspirations, and early activity and ambulation recommended. Here we have at the close of the war a verisimilitude of the treatment adopted with such brilliant results in the war to come.

During these intervening years we forgot the lessons that came out of World War I and were so ably stated in the official medical history of that war, and until the advent of the sulfonamides we continued to concern ourselves with the treatment of empyema by aspiration, closed drainage and irrigation by various methods, open thoracotomy, and the collapsing operations for chronic empyema. When this Association was founded empyemas both acute and chronic were so common and so much a part of the general surgical services that we debated whether or not they should be included in thoracic surgery.

Our civil injuries of the lungs and pleura were chiefly crushing or caused by bullets or stabbing with small instruments such as ice picks. The same applied to the heart and the pericardium and in this latter field significant contributions were made by Likin¹¹ and Bigger¹² who reported to the Association in 1935 and 1938. From his experience with a small group of patients with bullet wounds of the chest from the New York Police Department Weeks¹³ advocated prompt evacuation of the hemothorax and re-expansion of

the lung in order to assure early return to duty and avoid protracted invalidism. Also during this period we witnessed the enormous advances in anesthesia and the introduction of the sulfonamides so that when war came to us in 1942 we were far better equipped than we were in 1917. In addition and perhaps most important of all, our intern and residency systems were at a high stage of development with many young men well trained and ready to take over where their predecessors had left off. Unfortunately, however, although whole blood transfusions were freely used in civil practice we had neglected to read aright the significance of Robertson's work a quarter of a century earlier, and instead had been lulled into a sense of false security by false concepts of blood substitutes. Thus, as we entered World War II the whole emphasis was placed upon plasma and the so-called blood substitutes and no preparations had been made for the use of whole blood in quantity.

WORLD WAR II

We were catapulted into the war in three theaters, each with distinctly different types of warfare, which in turn brought their own peculiar problems and diseases. In all, however, the causative agents of wounds were similar to World War I except that high explosives were the causative missiles in nearly 80 per cent instead of 70 per cent as in World War I. Fortunately, preventive medicine was at its best, so that there were no epidemics in the camps at home nor did we have to contend with a world-wide epidemic of influenza. Pneumonia likewise had been on the decline for many years, and we had the sulfonamides as powerful therapeutic agents. From the start therefore we were not bothered by infection, but were able to focus our attention upon the proper surgical treatment of the wounds on an anatomic and physiologic basis with early closure and restoration to as nearly normal function as possible. It was realized from the start by the three Theater Consultants E. D. Churchill, E. C. Cutler, and W. B. Parsons, that the best prevention for infection in wounds was the proper initial surgery. It was recognized very early also that plasma was no substitute for blood, and it is a matter of record that the various hospitals in the overseas theaters used an ever-increasing amount of whole blood in the treatment of their casualties. At first this blood was obtained from the hospital personnel and also from the convalescing and ambulatory patients. Colonel Churchill was well aware of the necessity for whole blood available in large quantity, and it was due to his persistence of effort that the first blood bank was established in Naples which not only supplied fresh blood for the Mediterranean Theater but also cared for the needs of the Seventh Army during its invasion of southern France and until its supply was taken over by the European Theater in November, 1944.

As regards injuries and wounds of the chest under the guidance of Colonel Churchill attention became centered upon the lung rather than on the pleural space, the physiologic disturbances incident to wounding required prompt treatment sometimes in the most forward hospital unit, and with extensive surgery when necessary. Emphasis was placed upon adequate resuscitation, prompt re-expansion of the lung, and rehabilitation of the wounded man and in the communication zone chest centers were established for expert

reparative surgery and convalescent care, with the result that only about 11 per cent of those with thoracic wounds developed empyema^{14 16} The indications for formal thoracotomy at initial surgery were (1) continuing intra pleural hemorrhage, (2) wounds of the trachea or larger bronchi, (3) wounds of the mediastinal organs, (4) wounds involving the diaphragm, (5) presence of large foreign bodies in the pleura¹⁵ For the other wounds only a local débridement and aspiration of the pleura was performed Decortication was done later as indicated in the empyema group and in the small remaining group of noninfected clotted hemothorax cases Many in this Association contributed much to these brilliant results

In the European Theater after the invasion a similar regime was followed Chest centers were established in Great Britain under the direction of General Cutler, the Theater Consultant, and it was in one of these centers that Harken¹⁶ made his great contribution to cardiac surgery, the safe removal of intra cardiac foreign bodies

Although the final statistics of World War II have not yet been published, in 20,000 chest wounds treated up to 1944 the mortality was 8.1 per cent⁹ Perhaps the treatment of thoracic wounds reached its zenith from the spring campaigns of 1944 to the close of the war, because it was at about this time that penicillin became available in quantity, blood banks in the United States and in all theaters were well established, and in the personnel of the hospitals and the auxiliary surgical teams were a large number of well-trained surgeons who had profited not only by their experience at home, but were now also veterans in the surgery of the trauma of war It was with such a group that I found myself in the Seventh Army (Tables IV and V^{9 17}) In an analysis of over 55,000 wounds treated in the Seventh Army Hospitals from Oct 1, 1944, to May, 1945,¹⁷ the field hospital platoons treated 264 thoraco-abdominal wounds (5.3 per cent) and 1,675 thoracic wounds (33.6 per cent) In other words wounds involving the thorax comprised 38.9 per cent of admissions to our most forward hospital units On the other hand, the evacuation hospitals, and at times some of these were within a half mile of the field hospital platoon, rendered primary hospital treatment to 187 patients (0.4 per cent) with thoraco abdominal wounds and 2,645 (5.3 per cent) with thoracic wounds—5.7 per cent of their admissions This is an example of the proper proportion of the work load in these two types of hospitals The total number of thoracic wounds amounted to 4,320, or 7.8 per cent of our wounded Of the 1,675 direct admissions to the field hospitals, only 38.5 per cent required formal operative surgery, and 59 per cent were evacuated after resuscitation and aspiration to the evacuation hospitals, 2.5 per cent died before operation could be accomplished

As might be expected the mortality in these two field installations was entirely different For the field hospitals the mortality of the thoraco abdominal cases was 26.5 per cent (in the last 200 cases cared for by the Second Auxiliary Surgical group the mortality was 20 per cent) and 15 per cent in the evacuation hospitals with an over all mortality of 21.7 per cent This

TABLE IV PERCENTAGE OF WOUNDS ACCORDING TO CAUSATIVE AGENTS

WAP	BULLETS	HIGH EXPLOSIVE FRAGMENTS
American Civil	90 10	9 80
World War I		
British	38 98	60 70
American	28 06	71 62
World War II		
Russian	20 00	80 00
American	19 50	80 40
Fifth Army		82 67
Seventh Army		75 20
Korea		
UN		
Bullets mortars grenades	70 00±	

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TABLE V MORTALITY FROM CHEST WOUNDS

	NUMBER	DEATHS PER HUNDRED CASES
American Civil War		
World War I U.S.A.	20,607	27 8
World War II, U.S.A.	4,595	24 06
U.S. Seventh Army		5 2
Thoraco-abdominal	4,320	5 4
	451	21 7

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group comprised the most severe types of wounds usually multiple and often with large fragments from high explosive artillery shells. The purely thoracic wounds, however, in the field hospitals suffered a mortality of 87 per cent, and in the evacuation hospitals 3.3 per cent, an over-all mortality in the Army hospitals of only 5.4 per cent. As you will note, there has been no differentiation as to whether the wounds were penetrating, perforating, crushing or from blast, as this was not considered when tabulating our figures. These statistics are similar in the hospitals in the forward zones, both in the Mediterranean and European theaters in the last year of the war and show marked improvement over the earlier statistics up to 1944. At that time our men were receiving the maximum of fire from the Germans, from artillery, land mines, and from the air, with the causative agent 82.6 per cent high explosive missiles in the Fifth Army¹⁸ and 75.2 per cent in the Seventh Army.¹⁷ The chief contributing factors, I believe, to this really amazing record were first, the training and skill of the many surgeons involved, second, the excellent resuscitation afforded us with the fine judgment bred only by experience and soundness of knowledge of the physiological needs of the human body, third, the skilled and effective triage by the division clearing stations, fourth, the fluidity of personnel exchange and the availability of expert surgical teams, fifth, the Consultant system, and sixth, the antibiotics and other antibacterial agents. This is a complete about-face from World War I, when it was believed that the antibacterials and antiseptics were of ultra-prime importance

I have dwelt upon these results in the last year of World War II at some length. Although the official medical history of World War II has not yet been published by the Office of the Surgeon General, the whole conflict is fresh in the minds of many, and the many excellent articles that have appeared provide easy reference for the new generation of surgeons that has come upon the scene within the last six years. During this period also there has been far closer cooperation between the Officers of the Surgeon Generals and the civilian profession through the Society of Consultants of World War II, the Medical Advisory Council to the Secretary of Defense, the less formal surgical clubs arising from the war, liaison between the Surgeon Generals' Offices and the medical schools, the Committee on Medical Sciences of the Research and Development Board, and with the establishment of the Section on Military Medicine by the American Medical Association.

KOREA

Nevertheless, when the Korean conflict began in June, 1950, there was neither preparation, available personnel, nor the necessary medical units. Therefore, those in charge, particularly in Tokyo, had to make the best of an extremely difficult situation. The field hospital with its three platoons had been abandoned in favor of the Mobile Army Surgical Hospital (MASIH) which was constituted to accommodate priority I surgical patients with the concept that it should be placed adjacent to the clearing station, as had been the field hospital platoon. Its personnel consists of a fixed complement of about twelve Medical and three Medical Service Officers. The Commanding Officer, a Lieutenant Colonel, is listed as an experienced surgeon of Brigade. There were no auxiliary surgical teams, and for some reason these were no longer regarded in very high favor, as it was thought that the MASIH unit with its intrinsic personnel could handle existing situations without difficulty. The MASIH units were assigned to the Army Surgeon for attachment at the rate of one per division. These units, however, had never been assembled except on paper. Furthermore, as the war broke there was but one evacuation hospital in existence in the United States; hence the Surgeon of the Far Eastern command in Japan had to do his best with the meager facilities at hand. In addition to a lack of necessary units and the superb field medical organization that existed in 1944 and 1945, personnel had been pinned to the bone and many officers were in or just finishing residencies in the re-educational program of the Army. This happily provided a small nucleus of well trained capable specialists in the various branches of the medical profession.

Korea itself presented a special situation. It is a narrow peninsula with several high mountain ranges running north and south and deep valleys in between. The mountains vary from a few hundred to seven or eight thousand feet in height and are steep and in large measure barren. There are two railroads up the peninsula: one through the large central valley and one along the east coast. The north and south roads followed a similar course and were not good; both were largely the work of the Japanese during their occupancy. There were almost no lateral communicating roads due to the terrain itself.

Thus forced all military traffic over these few roads and the two railroads, which soon began to deteriorate under the strain. In order to meet this desperate situation in the best manner possible, and in view of the fact that the United Nations Forces were in a movement of rapid retreat something had to be done in a hurry. Therefore, M.A.S.H. units were hastily organized and blown up into small mobile evacuation hospitals as an emergency measure so that each was theoretically able to care for two hundred patients although at times they had six hundred. All patients passed through them on the way to the rear thus creating a bad situation from the start in that these hospitals had to perform triage, priority I, and priority II surgery.^{18 19} Obviously triage suffered somewhat and priority II surgery suffered the most (Table VI)

TABLE VI REPORT OF M.A.S.H., JULY 9 DECEMBER 31, 1950

Admissions		8,577
Patients operated		1,516
Number of surgical procedures		2,280
Laparotomies	221	
Thoracotomies	60	
Amputations	59	
Head cases	10	
Debridements	1,173	
Number of pints of whole blood used		1,298
Deaths		112

Modified from Bowers, W. C., Merchant, F. T. and Judy, K. H. The Present Story on Battle Casualties from Korea. Surg. Gynec. & Obst. 93: 529, 1951.

At times great confusion prevailed, but with the able support of the Air Force and the Navy an excellent evacuation service was soon in operation and hospital ships were dispatched to Pusan to function as stationary evacuation and station hospitals. The required personnel were gathered from the small group within the Regular Army, from those who volunteered for extended duty and certain doctors who were called in on an involuntary basis either singly or as members of Reserve Units. The hospital system in Japan was expanded at the same time and ably though thinly staffed and, in order to assure the best possible care in the earlier months, emphasis was placed on evacuation. This was both easy and costly, however. Inasmuch as the planes were returning empty to Japan, Hawaii, and the United States, there was plenty of room for the wounded and ill, and unquestionably in the early days better care was available in those places than in the crowded hospitals of Korea. But at the same time many were evacuated who could easily have been kept either within the combat area or the forward part of the zone of communication had proper hospitalization been available. Selective rather than total, evacuation with adequate combat medical organization and communication zone facilities is essential to conserve man power and morale within the fighting area. Mindful of these facts, and knowing also the magnificent record that had been established by the Medical Department in Korea in spite of what seemed at one time almost overwhelming obstacles I arranged with the Surgeon General for a visit in December, 1951 to try to learn in what way we could be of further assistance and so report to you.

I traveled from the Battalion Aid Stations of the Marines through the hospitals of Korea, Japan, and Okinawa. First, I should like to give unserved praise to the morale of all the troops, then superb logistical support, and the extraordinary work of the engineers in improving the roads and in cutting new roads so that now almost all, even those winding through the mountains, are two-lane highways for trucks. There was excellent spirit in all the United Nations Medical Units in all Corps areas, East and West, and the support and care rendered by these organizations, not only our own three Services, but also Norway, Sweden, Denmark, and India merit the highest praise. As you know, the front was quiet in December, and the surgeon of the Far Eastern Command was in a quandary as to what to do with his hospitals, which were admittedly out of position at that time with the MASH units about twenty miles behind the lines, because should an armistice be agreed upon emphasis would be placed on caring for the returning American prisoners. On the other hand, if war were to continue, some rearrangement of hospitals and additional personnel would be required.

Korea is a desolate and lonely place. From the standpoint of our medical units, each is more or less isolated, but the personnel are brought together once a month for medical meetings in each corps area. These have been a godsend, as communication between the units is difficult. There is little emotional drive to create enthusiasm, and all the men are looking forward to the time when they can leave Korea. This personnel has been held to a bare minimum, in December there were only 49 experienced general surgeons in fifty hospitals in the entire Far Eastern Command, and 13 experienced orthopedists and 2 neurosurgeons. These included the consultants both from General Headquarters and the Eighth Army.²⁰ The remaining 122 general surgeons were interns and men partly through their residencies, together with some who had been in general practice with an interest in surgery. The personnel of the MASH units was so scant that there was little attempt at organization into teams. The problem was made still more difficult by the frequent shift in both officers and enlisted men. Both the theater and army consultants are doing almost superhuman work in covering the hospitals and instructing, attending medical meetings, and keeping up with their reports. In addition, they travel each month with the visiting Consultants from the States. The young men in all the hospitals are eager for informal discussions and talks about their work and problems. Some in the hospitals in the communication zone were requesting service in the Eighth Army units. This so impressed us that a policy was established whereby all new arrivals would first have service in the hospitals in the rear so as to indoctrinate them in the problems of forward surgery and the mistakes that might be made. After a period of several months they are then to be sent to the Eighth Army and its installations. It is believed that with this training they will do even better work in the combat zone. It has not been possible to adhere to the original organization of the MASH units, with the result that some of the commanding officers are not surgeons at all which thereby diminishes by one the already limited surgical personnel. Also some are not interested in teaching the

younger men on their staffs. The greatest deficiency that both my successor²¹ and I found was ignorance of the pathology of the high-velocity wound and the reason and necessity for careful debridement. By and large the major wounds, such as those of the abdomen and chest were well cared for although here again there was a failure to realize and also to absorb from the teachings of the overworked consultants the fundamental surgical principle that each compartment of the body must be cared for in wounds involving more than one area such as thoraco abdominal injuries and wounds involving the buttocks, rectum and bladder. This of course resulted in complications that presented extraordinarily difficult problems. These were being so well handled by the surgeons in the rear hospitals that it was evident that they were frequently having to correct the deficiencies of the initial surgery.

It is not a function of this paper to go into the surgical specialties but in passing I should like to pay tribute to the really outstanding performance of the neurosurgeons who already have two teams of doctors, nurses, and enlisted men with a small amount of housekeeping equipment in the forward areas and the Neurosurgical Center in Tokyo. I have never seen a group of patients so well thoughtfully and considerately cared for. As to our own work there is a Thoracic Center in Tokyo Army Hospital which was well supported by the Chief of Surgery and ably conducted by the surgeon in charge.

I was somewhat disappointed in the attitude of some of the younger men in the forward hospitals that their time was being wasted and they were bored at being forced to concern themselves with the surgery of trauma. This was brought acutely to mind by one of my own residents who was about halfway through his residency and had been with a forward unit for eight months. He told me that he considered his entire time spent with that unit which had always been extremely active as just so much time wasted as regards surgery. I believe this attitude is the fault of us at home. Perhaps we should pause and consider if the definition of surgery as given by Theodoric in the thirteenth century is not just as true today possibly more so, as it was then. He said surgery consisted of three parts: (1) the joining together of broken parts, (2) the separation of parts unnaturally joined, and (3) the removal of whatever is superfluous.²² Have we paid too much attention to the last two and neglected the first? Certainly the desire of all young surgeons today is to remove and remove until there is not much left because somehow or other it seems to be more interesting to subtract than to restore. But is this a true criterion of the surgeon? I would remind them of the wisdom Dr. Nathan Green offered in his presidential address in 1924: "It is of a subtler quality of the mind to know when and when not to operate than to know the technique or procedure."²³

Quite apart from battle casualties, in the first year and a half in Korea there were 22,000 road accidents.²⁴ In December last we had our one millionth death from the automobile and during 1951 93,000 were killed in accidents throughout this country and 9,100,000 were injured, at an economic loss of eight billion dollars.²⁵ Fatalities were four thousand greater than in 1950 and motor vehicle accidents now account for about thirty-five thousand deaths

a year. On the other hand, our one millionth death in war since the Battle of Lexington occurred on Sept 4, 1951, in Korea²⁶. With the exception of the formation of the Association for Trauma, which is becoming increasingly important, and committees of the American College of Surgeons and National Research Council, we tend to negate the treatment of trauma in our better teaching hospitals and not infrequently men will pass through an entire residency in general surgery without ever having to face the problem of the seriously injured patient. This was brought out somewhat forcibly two years ago at the oral examinations for the American Board of Surgery when two candidates presented themselves before another examiner and me. Each had a patient who had been struck by an automobile, and in each instance there was a fractured pelvis, a common enough injury on the roads or in industry. Both of these young gentlemen had graduated from good schools and had their residencies in teaching hospitals, still, it required a good deal of prodding and prompting on our part to get them to recommend any other treatment than that they would call an orthopedic consultation. The urological aspect did not enter their minds and, when asked about the possibility of bladder injury, why of course a urologist would also be called in consultation. When pushed to the point of what they would do themselves if the facilities of the medical center were not available, they each decided they would merely put a drain down to the opening in the bladder were it injured. I have recently had a conversation with another young man who had spent nine years in a residency in another of our teaching hospitals on the general surgical service, and when the same question was put to him as to how he would treat a seriously injured patient he said he would immediately ask for a consultation. In other words, it has become rather "old hat" to pay attention to reconstruction of the human body in anywhere nearly the same manner as to the "radical cure" of disease, with its necessary eviscerations and dismemberments. I do not wish at all to maintain that this latter is not a most important part of our teaching, but I also believe firmly that we should reorient our teaching of surgery so as to place increased emphasis on trauma and its various causes and pathology and treatment. I do not mean that one person should necessarily assume the entire responsibility, but I believe that everyone passing through our residency systems should have sufficient knowledge so that he can handle a given situation from the standpoint of initial surgery until he can get help. This means an understanding of the fundamentals of surgery, resuscitation, and restoration of anatomy and physiology in the different parts of the human body. As we are in an age of war, we should teach the pathology of trauma and healing of wounds, and the importance of comprehending the different types of causative agents and their sequelae.

War and trauma unfortunately will not decrease in the foreseeable future. It becomes, therefore, our responsibility to teach and emphasize the surgery of repair and reconstruction just as much as we teach the surgery of pathology and disease so that when our interns and residents enter military service or encounter disaster they will not be perplexed and bewildered at the devasta-

tion wrought on human frames by man and his machines, but will be prepared to treat their torn and wounded fellow men with sound knowledge of the fundamental principles of the initial surgery of wounds

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FIBROSARCOMA OF THE BRONCHUS

REPORT OF THREE CASES DIAGNOSED BY BRONCHOSCOPY AND TREATED BY RESECTION

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INTRODUCTION

PRIMARY fibrosarcoma of the lung is a most uncommon lesion, and neoplasms of this type of proved bronchial origin are extremely rare. In a recent review, Black¹ was able to find only five cases in the entire medical literature which he considered to fulfill the criteria of primary fibrosarcoma of the bronchus. He added one case of his own. In the same issue of the JOURNAL in which Black's case was reported, Carswell and Kiaeft² described an additional case which was diagnosed by bronchoscopy and treated by pneumonectomy. Those authors reported having found six previously described well-authenticated cases of primary bronchial fibrosarcoma. Finally, Curry and Fuehs³ have placed on record the case of a rapidly growing fibrosarcoma expectorated by a 13-year old girl, it was believed to be of bronchial origin. However, the patient has survived for over four years, and the primary site has not been conclusively established.

Reports of pulmonary sarcoma are commonly encountered in the older medical literature, but it appears that a vast majority of the tumors so described were either carcinomas or lesions of lymphatic origin. As recently as the turn of the century, carcinoma of the lung was considered a rare tumor. For example, Rolleston and Trevor,⁴ in 1903, found only two bronchiogenic carcinomas in 3,983 autopsies at St. George's Hospital. They invited attention to Passler's⁵ figures which included sixteen primary carcinomas of the lungs and bronchi among 9,246 post-mortem examinations at the Breslau Pathological Institute. Of the patients studied by Passler, 1,000 died of malignant disease, 870 of them from carcinoma, but the percentage of lung cancer was found to comprise only 1.84 per cent of the primary epithelial tumors, and only 1.6 per cent of the total group of malignant disease in the series. Their own experience, and a review of the literature to that time, prompted Rolleston and Trevor to conclude that 'primary malignant disease of the lung proper, whether of the body or root, is of the nature of sarcoma' and their belief appears to be fairly representative of the medical opinion at that time. Discussing reports in the medical literature of primary pulmonary carcinoma, those authors stated that

the conclusion is forced upon us that some of the older writers have wrongly named their cases. It was their feeling that most of the reported cases were in fact sarcomas.

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Present experience indicates that the exact opposite situation actually prevails. It is now well established that primary bronchiogenic carcinoma is one of the most common of all internal cancers, being rivaled only by carcinoma of the stomach as the most frequently encountered malignant neoplasm in the male.⁸ Conversely, increasingly precise pathological studies have clearly shown primary sarcoma of the lung to be a variety of tumor seen only on the rarest occasions. Although it is generally accepted that there has been a real, rather than merely an apparent, increase in the incidence of primary carcinoma of bronchial origin during the present century, it is almost the unanimous opinion among present-day pathologists that a great majority of the large number of sarcomas of the lung reported in the earlier literature were in reality tumors of epithelial origin. Edwards⁹ commented upon the rapid disappearance from the literature of reports of primary pulmonary sarcoma in the 1920's, and attributed this to Bernard's emphasis on the fact that more meticulous study would enable pathologists to differentiate the sarcomas from highly immature epithelial tumors. Adler⁷ listed ninety cases of lung sarcoma in his book published in 1912, but could authenticate only a small minority of them. Reviewing his material, it is found that a lack of adequate pathological description makes it impossible to accept a single case of Adler's as representative of primary bronchial fibrosarcoma. Furthermore, Adler stated that he had never personally seen a primary sarcoma of the lung. The rarity of primary sarcoma of the bronchus in the experience of present observers is attested by the fact that Mallory,¹⁰ in 1936, in reporting a case of spindle-cell sarcoma of the lung, believed to be primary, stated that it was the first such case encountered at the Massachusetts General Hospital in over 8,000 post-mortem examinations performed there up to that time. Three years later, Ellis¹¹ described a case of primary fibrosarcoma of the lung seen at the University of Kansas Hospital, and noted that it was the first found at that institution in 7,272 autopsies. Clinical findings are in close agreement with the necropsy statistics just given. For example, Oschner, DeBakey, Dunlap, and Richman¹² reported 548 cases of primary pulmonary malignancy, of whom 195 underwent resection. In that series, only one sarcomatous lesion, described as a neurofibrosarcoma, was encountered. This experience closely parallels that described in other clinical reports.

LITERATURE

Ball¹³ reviewed the literature on primary pulmonary sarcoma in 1931 and found reports of only fourteen cases which he considered to be adequately authenticated. He reported an additional case of his own. Six of the cases accepted by Ball, however, were "round cell sarcomas" and, therefore, probably should be classified in the lymphoblastoma group. A seventh case, described as "fibrochondroadenoma with osteoid tissue" clearly cannot be accepted in the group of bronchial fibrosarcomas. Of the remainder, only the cases of Boschowsky¹⁴ and Ball presented convincing evidence of being truly representative of fibrosarcoma. In each of these cases the pulmonary involvement was so extensive that it could not be determined with certainty whether the tumor arose

in a bronchus or from the pulmonary parenchyma or pleura. This also applies to additional cases reported by Mallory,¹⁵ ¹⁶ as well as those of Scheidegger,¹ Klempeier and Rabin,¹⁸ Lyssunkin,¹⁹ and Stohl and Sachs.²⁰ The cases included in these reports were clearly fibrosarcomas of the lung, but again their wide spread involvement of a lobe or an entire lung makes it impossible to classify them as primary fibrosarcomas of bronchial origin, their primary site must remain conjectural. The two cases recently recorded by Gray and Whitesell,²¹ both pedunculated tumors attached to the lung only by a slender pedicle, while true fibrosarcomas of low-grade malignancy were almost certainly not primary in a bronchus. It appears likely that they arose from the pleura, or, possibly, from the pulmonary parenchyma. The case reported by Steward²² showed a predominance of spindle cells and could possibly be classed as a fibrosarcoma, but some small round cells were also seen, and the author described the case as a "mixed cell sarcoma." That patient had a large mass involving the entire left lower lobe, and the exact site at which the tumor arose could not be determined. Rosenblum and Gasul,²³ in a report lacking in pathological detail, recorded a case described only as "sarcoma" encountered in an infant. They referred to the reports of Bjoirsten²⁴ and Lehnhoff,²⁵ who described *round-cell sarcoma* in children, aged 2 and 3 years, respectively. Rosenblum and Gasul reviewed the pediatric literature and found eleven cases of sarcoma occurring in older children. The conclusion appears warranted that all of these tumors were of the round cell variety. Stout²⁶ encountered only four cases of fibrosarcoma of the lungs, pleura, or bronchi in the Laboratory of Surgical Pathology at Columbia University during the years 1907 through 1946. During the same period, a total of 362 fibrosarcomas in all locations were seen. Johns and Sharpe²⁷ reported a well authenticated fibrosarcoma of the bronchus in an 18-year-old boy. Death occurred from respiratory complications before treatment could be instituted. Autopsy showed almost complete replacement of the right lung by tumor. A polypoid tumor mass extended along the right main stem bronchus and into the trachea.

BRONCHOSCOPY

Bronchoscopic examination and biopsy has for years been standard procedure in the diagnosis of tumors of the lungs and bronchi. Although supplemented in recent years by studies of exfoliative cytology, it remains by far the most important single method of investigation by means of which the exact pathological nature of a suspected bronchial lesion may be definitely determined preoperatively. In spite of the widespread employment of bronchoscopy, with the diagnosis of countless cases of pulmonary carcinoma having been established by that means, the number of cases of sarcoma of the lung diagnosed by bronchoscopic biopsy is extremely small. In 1939, Baum²⁸ and his associates reported the case of a 29 year old white woman who showed a polypoid lesion of the right main stem bronchus. The diagnosis of spindle cell sarcoma was established by bronchoscopic biopsy. Those authors reviewed the literature, and could find only one such case diagnosed bronchoscopically, that of Herrnhiser²⁹ (1935), although Jackson,³⁰ in 1930, mentioned two endobronchial

spindle-cell sarcomas diagnosed by bronchoscopic biopsy Jackson's cases were neither illustrated nor described in detail, and no statement was made as to whether they were considered primary in the bronchus or metastatic lesions. No follow-up was given. In 1911 Kahler³¹ described before the International Laryngorhinological Congress a patient upon whom direct tracheoscopy showed a mass completely obstructing the right main bronchus. The patient subsequently coughed up a fragment of tissue which was diagnosed histologically as myxosarcoma. The lesion was treated by cauterization through the bronchoscope, but the tumor recurred. The clinical report was brief and was not illustrated by photomicrographs of the tumor. Additional cases diagnosed by bronchoscopic biopsy have been reported by Mallory¹⁸ (1938), and Pollak and associates³² (1941), Lewis³³ (1946), Black¹ (1950), and Carswell and Kraefitz² (1950). Reports of lymphosarcomas and other tumors of the round-cell type, such as those of Vinson³⁴ and Jackson,³⁰ have been excluded in that it is felt that these tumors arise primarily in the mediastinal and peribronchial lymph nodes, and involve the bronchi and pulmonary parenchyma only secondarily.

TREATMENT

The meager experience which has been acquired in the management of patients suffering from primary fibrosarcoma of the bronchus makes it clearly impossible to formulate a therapeutic program which can be statistically shown to be the most rational and effective method of dealing with these lesions. The few cases that have been reported have been treated in a variety of fashions from the most conservative to the most radical. The 13-year-old girl reported by Curry and Fuchs³ received no treatment, since the exact source of origin of the expectorated bronchial fibrosarcoma was never determined. She has been followed for over four years and shows no evidence of recurrence. The case of Pollak and associates³² was treated by bronchoscopic removal and fulguration. The patient was well at the end of two and one-half years. Baum and co-workers²³ treated their case by repeated bronchoscopic removal and massive irradiation. The patient received a total of 18,630 r units over a two and one-half year period. Death occurred two years and nine months after treatment was first instituted, post-mortem examination established pulmonary tuberculosis as the cause of death, and thorough examination of the affected lung, including careful study of the previous site of the bronchial tumor, showed no evidence of neoplasm. The case of Lewis³³ was managed in a similar manner, after bronchoscopic removal of the visible tumor his patient received 6,353 r units of irradiation. Two and one-half years later the patient was clinically well, and his chest roentgenogram was negative. Heinheiser²⁹ reported a complete cure by irradiation therapy. The use of radon seeds and electrocoagulation have been suggested for this condition. The case of Herzmans³⁵ was treated by lobectomy. There was no sign of recurrence of the tumor thirteen months later. The lesion in the case reported by Davis,³⁶ also removed by lobectomy, was obviously metastatic in character, and no conclusions can be drawn from the behavior of his patient. Sweet³⁷ did a right lower and middle lobe lobectomy upon a patient with a large solid tumor, shown on microscopic examina-

tion to be a fibrosarcoma which involved the pulmonary parenchyma in a very extensive manner. Although thought to be primary in the lung, the non metastatic character of the lesion was not definitely established. Connection with a bronchus was not cited, and one may assume that the widespread involvement of the lung made determination of the exact site of origin of the tumor impossible. The neoplasm recurred two years later in the thoracotomy incision, and there were metastases to the spine. In the case of primary fibrosarcoma of the bronchus reported by Mallory,¹⁵ death from intrapulmonary hemorrhage occurred during exploratory thoracotomy performed by Churchill.¹⁶ Right pneumonectomy had been planned. In discussing the case, Churchill suggested local bronchoscopic removal prior to pulmonary resection in order to clear the obstructed bronchus and thus make the lung more manageable at the time of definitive surgery. In a second case at the Massachusetts General Hospital, Churchill¹⁶ removed the left lung for a large sarcoma of spindle-cell type which involved the pleura as well as the parenchyma, and in addition was adherent to small portions of two ribs. Segments of the two ribs were removed with the lung. The origin of this tumor is therefore uncertain, Mallory felt that it was probably primary in the lung. Graham performed a left pneumonectomy in the case reported by Black¹, palliation was achieved for about six years, but the patient died of mediastinal recurrence six years and seven months later. Carswell and Kjaeff² also performed a left pneumonectomy in their case. The operation was done in January, 1948, and the patient was free of symptoms and showed no evidence of recurrence at the time of their report seven and one half months later. Harrington¹⁸ performed pneumonectomy in six cases described as "sarcoma." Five of these patients died within two and one half years, while the sixth was living without evidence of recurrence five years later. Of twenty-one "coin lesions" treated by resection by O'Brien, Tuttle, and Gerkaney,¹⁹ one was diagnosed as "sarcoma." Details of the case are lacking. The patient died nine months later of a ruptured intracranial aneurysm. Complete post-mortem examination showed no evidence of recurrence or metastasis of the lesion. Edwards⁸ reported three cases as "sarcoma of the lung," but the microscopic pathology was not described. Two were described as ossifying, and one lay at the periphery of the lung. One patient, who was considered inoperable, was treated by irradiation and was alive and well two and one half years later, one died following operation, and the third survived in good health for at least two and one half years following lobectomy. Three of the four cases seen by Stout¹¹ were followed, and all died of their tumor in less than one year. One of the three had proved metastases.

CASE REPORTS

CASE I—A No 472571. This 39 year old white man was admitted to the University Hospital Ann Arbor Mich on Dec 18, 1940, complaining of a chronic cough productive of two to four tablespoonful of whitish sputum daily, shortness of breath, and pain beneath the left scapula. He stated that chest roentgenograms taken elsewhere in June, 1939, had shown a left intrathoracic tumor. It gave rise to no symptoms, and observation was advised. Chest roentgenograms in November, 1939, showed no essential change in

the size of the lesion. Further watchful waiting was suggested. Roentgenograms in December, 1940, showed the tumor to be approximately four times its original size. There was also a left pleural effusion. Because of the rapid growth of the mass, hospitalization was urged.



Fig 1—(Case 1 A.A.) Posteroanterior chest roentgenogram taken Dec. 18 1940 at the time of the patient's admission to the University Hospital. In this view the huge tumor obscures the lower two thirds of the left hemithorax. Its rounded superior border is clearly shown but its inferior margin cannot be defined.

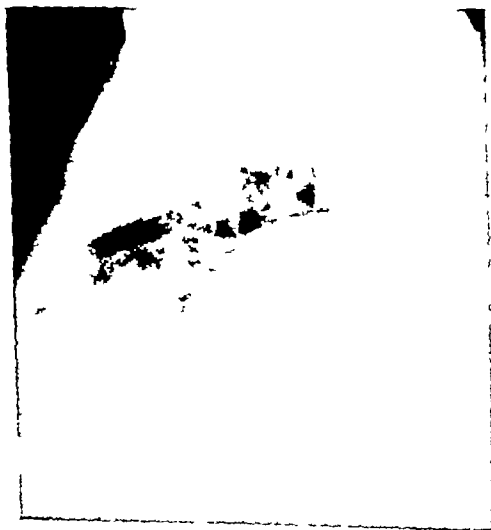


Fig 2—(Case 1 A.A.) Lateral chest roentgenogram taken Dec. 18 1940 showing the tumor occupying the middle and posterior portion of the left lower chest.

Physical examination upon admission to the University Hospital was essentially negative, except for the signs of consolidation in the lower left chest. Routine laboratory studies were normal.

Posteroanterior chest roentgenograms (Fig 1) showed complete obliteration of the lower two thirds of the left thoracic cavity. In the lateral film (Fig 2) the lesion was

seen to occupy a position in the middle and posterior portions of the chest. In this view it had a diameter of 5 centimeters. Chest films taken elsewhere approximately one year previously were reviewed. They showed a large, rounded, smoothly outlined, homogeneous shadow lying in the middle third of the left lung field as seen in the posterior anterior projection. A companion left lateral chest roentgenogram (Fig. 3) showed the tumor to be centered at the junction of the middle and posterior thirds of the hemithorax. Its posterior margin lay on a plane with the anterior borders of the vertebral bodies. These observations showed that the mass lay within the left lower lobe.

The patient was bronchoscoped by Dr. John Alexander on Dec. 18, 1940. A lesion was seen in the proximal portion of the left lower lobe bronchus, and this was biopsied. The following histologic report was made by Dr. C. A. Weller: "Beneath the bronchial mucosa there is a neoplasm composed of spindle cells with relatively scant cytoplasm. A spindle cell fibrosarcoma having characteristics which indicate origin in a neurofibroma."



Fig. 3.—(Left Lateral View) Left lateral chest roentgenogram, in which, in November, 1939, showing the homogeneous, rounded, smoothly outlined, dark shadow of the tumor in the middle and posterior thirds of the chest. A vertical anterior-to-posterior roentgenogram of the same field also showed the mass to be situated in the approximate center of the left lower lung field.

Left pneumonotomy was performed by Dr. John Alexander on Dec. 21, 1940. A huge tumor filling the lower three-fourths of the left hemithorax was found at operation. The upper lobe of the lung was collapsed, only the apical segment being preserved and expanded.

The left lung and tumor weighed 1,140 gram. The tumor was soft, gelatinous, and white. The histologic diagnosis by Dr. C. A. Weller was "Spindle fibrosarcoma of the lung."

The patient's postoperative course was essentially unremarkable except for a slight wound-healing problem. He was discharged on Jan. 1, 1941, with a chest tube in place and a drainage tube.

Postoperative treatment consisted of 100 mg. of hydrocortisone daily. He remained stable for six months, but then developed a cough and sputum production. A chest x-ray showed a large, rounded, homogeneous shadow in the middle third of the left lung field, similar to the one seen in the preoperative roentgenogram. This was followed by a series of chest x-rays showing the tumor growing in size.

*The patient was treated with 100 mg. of hydrocortisone daily. He remained stable for six months, but then developed a cough and sputum production. A chest x-ray showed a large, rounded, homogeneous shadow in the middle third of the left lung field, similar to the one seen in the preoperative roentgenogram. This was followed by a series of chest x-rays showing the tumor growing in size.

Chest examination revealed the findings incident to a left pneumonectomy. Ophthalmologic examination showed paralysis of the lateral rectus, O.D., vitreous opacities, O.S., right convergent strabismus, paralytic. The patient was referred to the Department of Neurosurgery where the opinion was expressed that his symptoms were due to a general increase in intracranial pressure from cerebral metastases.

The patient's hospital course was steadily downhill. He required liberal opiates for relief of severe leg pain. There was intractable nausea and vomiting. He was discharged on Aug. 23, 1941. It was the clinical impression at that time that he was suffering from recurrent metastatic fibrosarcoma, and that the prognosis was hopeless. He died at his home in the fall of 1941, approximately ten months after left pneumonectomy. Post mortem examination was not done.



Fig. 4—(Case 1 A.A.) Photomicrograph of the tumor. This lesion showed considerable variation in its microscopic appearance in different areas.

Comment—The tumor in this case was so large and the pulmonary involvement so extensive that it was not possible to determine with certainty the exact origin of the tumor. However, it was proved by bronchoscopic biopsy to have involved a major bronchus. Whether it was primary at that site might well be debatable. Clinically and microscopically, the lesion in this case was indistinguishable from the neoplasms in the two succeeding patients in this report. Serial chest roentgenograms clearly showed that the tumor arose within the substance of the left lower lobe. In passing, one might once again point out the extreme danger of the policy of "watchful waiting" as applied to intrapulmonary lesions which are thought to be benign. This case exemplifies the wisdom of Alexander's dictum that "most intrathoracic neoplasms are not extrapulmonary and benign but intrapulmonary and malignant."⁴⁰

CASE 2—M. C., U. H. I. No. 651010. This 62-year-old white woman entered the University Hospital on Dec. 2, 1948, complaining of chronic cough of six months' duration. Her sputum had recently increased in amount and become more purulent in character. During the few weeks preceding admission she had raised small quantities of blood on several occasions. There had been slight weight loss and increasing sense of fatigue.

Physical examination showed slightly diminished expansion of the right chest when compared with the left. Tactile fremitus was increased over the right lower chest posteriorly and bronchovascular breath sounds were heard anteriorly and posteriorly over the right lower chest. Chest roentgenograms showed evidence suggestive of atelectasis involving the right basilar segments (Fig. 5).

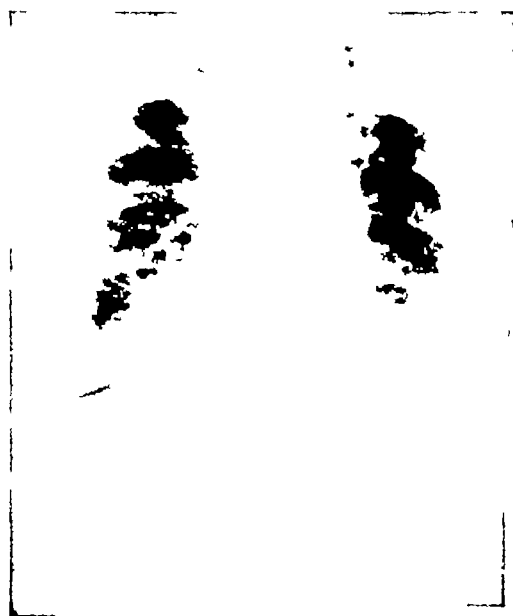


Fig. 5.—Chest roentgenogram, M. C., U. H. I. No. 651010, taken on Dec. 2, 1948, showing evidence of atelectasis of the lower segment of the right lower lobe. A lateral film indicates that atelectasis.

Br. ch. bx. was performed by C. F. S. on Dec. 12, 1948. The right main stem bronchus was normal but produced no sputum. A 1.5 cm. nodule, firm to a point, was palpable in the lower lobe. The area of the apex of the lower lobe was firm.

meters Superiorly, it extended into the main lower lobe bronchus thence upward to terminate opposite the right upper lobe orifice En route it sent out projections or pseudopods which partly or wholly occluded the orifices of the other three basal segmental bronchi, the bronchus to the superior segment of the lower lobe and the middle lobe bronchial orifice (Fig 6) The histologic diagnosis (by Dr C A Weller) was "Pleomorphic spindle-cell fibrosarcoma" (Fig 7)



Fig 6—(Case 2 MLC) Photograph of the gross specimen. The lung has been sectioned along the major bronchi. The intrabronchial tumor yellowish white in color in the fixed specimen, formed an almost perfect cast of the bronchi in which it lay and sent out pseudopod-like projections into adjacent bronchi. It was actually attached by a very small base no more than 4 to 5 mm. in diameter in the midportion of the medial basal branch bronchus

The patient's postoperative course was uncomplicated, and she was discharged on the fourth postoperative day. Chest roentgenograms eleven months later showed that she was developing overdistention of her left lung (Fig. 5). In November, 1950, a right posterolateral thoracoplasty was performed in order to shift the mediastinum back to the midline and to arrest the compensatory emphysematous changes in the left lung. At the time of that admission there was no evidence of mediastinal or other recurrence of her neoplasia.

The patient's final admission was on March 29, 1950. She complained of pain in the occiput, the left shoulder, the left ear, and the left upper neck. She had recurrent developed progressive paresis of the left arm. Examination revealed cervical metastases spine films showed narrowing of the intervertebral foramina between C4 and C5, and between C5 and C6 on the left. A small hard nodule was palpable in the right axilla. This nodule was removed, and on histologic examination showed spindle cell fibrosarcoma, apparently metastatic. A neurological consultant felt that there was involvement of the



Fig. 5. Chest roentgenogram, November, 1950, showing overdistention of the left lung.

metastases which occurred in this case, first becoming evident fifteen months following pneumonectomy, were probably via the blood stream. This case illustrates well the malignant nature of these lesions, and shows that distant metastases can occur even in the complete absence of local invasion.

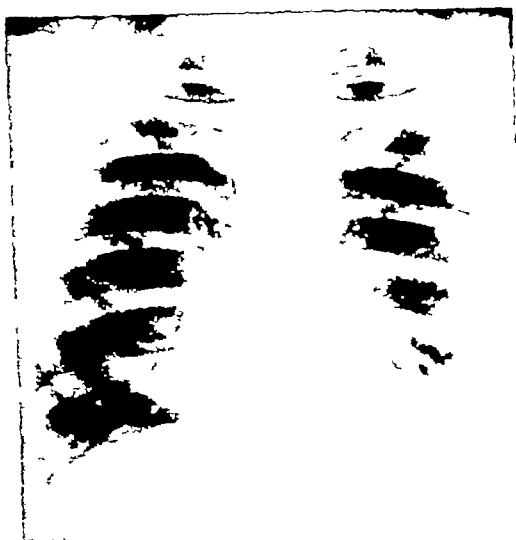


Fig 9—(Case 3, BS) Posterior anterior chest roentgenogram taken May 9 1949 showing suggestive evidence of partial atelectasis of the right middle lobe. Note elevation of the right hemidiaphragm.



Fig 10—(Case 3, BS) Lateral chest film taken May 9 1941 which confirms the impression of partial atelectasis of the right middle lobe. The shrunk semiopacified middle lobe is well shown in this projection.

CASE 1—B. S. U. H. Reg. No. 477699. This 41-year-old white woman was admitted to the University Hospital on May 9, 1949. She developed chills, fever, and malaise in December 1948. Chest examination revealed a pneumonic process in the right middle lobe. She had had several previous episodes of pneumonia, and during an attack in January 1949, a chest roentgenogram showed findings consistent with atelectasis of the right middle lobe. She was bronchoscoped in February 1949, and a tumor was seen in the proximal portion of the right lower lobe bronchus situated just distal to the middle lobe bronchial orifice. Biopsies were taken and on histologic study (by Dr. C. V. Welby) were interpreted as showing a moderately cellular fibroma.

The pertinent physical findings were confined to the chest. The lung fields were normal on auscultation, but on percussion a triangular area of dullness in the right midlung field anteriorly was noted. Tactile and vocal fremitus were markedly increased in this region.

Radiographic examination of the chest showed atelectasis or partial atelectasis of the right middle lobe (Figs. 9 and 10).



The patient's postoperative course was uncomplicated, and she was discharged on the twelfth postoperative day. She has been carefully followed since her discharge from the hospital, and when last examined on June 12, 1950, fifteen months after her pulmonary resection, she remained clinically well. There was no evidence on roentgenogram (Fig 12) or physical examination of recurrence of her neoplasm.

Comment—This case appears to represent the smallest bronchial fibrosarcoma on record to be diagnosed by bronchoscopy and treated by resection. There is every reason to offer a favorable prognosis. This case illustrates the serious significance of recurrent bouts of pneumonitis, particularly when there is associated atelectasis. It also shows the extreme value of careful bronchoscopic examination in that type of case.

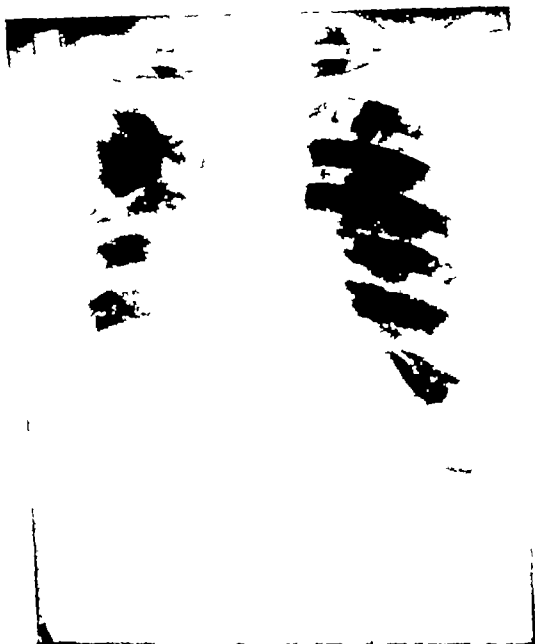


Fig 12—(Case 3 B.S.) Chest roentgenogram made May 8, 1950, one year after right middle and lower-lobe lobectomy for fibrosarcoma of the bronchus and three months after resection of a segment of the right for suspected recurrence. On pathological study that lesion proved to be a benign chondro osteoma.

DISCUSSION

Although it has been repeatedly stated that fibrosarcoma of the bronchus, in common with lesions of this type encountered elsewhere in the body, is a slowly-growing tumor, histologically well differentiated, spreading principally, if not exclusively, by local invasion, our own experience and a review of all well-verified cases reported in the literature lend poor support to that viewpoint. While exceptions have been noted, this entity has proved itself to be a highly fatal condition, usually causing death of the patient within two years or less from the time that symptoms first appeared. Case 2 illustrates that distant metastases can and do occur. Though not proved, similar spread doubt-

less occurred in Case 1. Despite the occasional success reported in the treatment of fibrosarcoma of the bronchus by conservative means, such as local excision through the bronchoscope followed by irradiation, it is impossible for me to subscribe to any such therapeutic regimen.

In the days when all intrathoracic operations constituted a grave risk there was perhaps some justification for avoiding resection therapy. At the present time, however, when resection of the lung can be undertaken with relative safety, hilly measures in attacking malignant disease of the pulmonary system scarcely seem defensible. It appears hardly appropriate to compare fibrosarcomas of the bronchus with bronchial adenomas in relation to therapy. I agree with Black,¹ who concluded that, in spite of the apparent success of endobronchial treatment in three reported cases, the therapy of bronchial fibrosarcoma must be by pulmonary resection in order to insure removal of all of the neoplasm. It is quite true that adenomas of the bronchus were long held to be benign lesions and on that basis it was felt that bronchoscopic removal constituted adequate therapy. Added experience showed, however, that these tumors often assume a dumbbell shape with much of the tumor invading the pulmonary parenchyma and inaccessible to the bronchoscope. Furthermore it has been well established that these are low grade carcinomas and that distant metastases sometimes occur. Pulmonary resection is being ever more widely accepted as the procedure of choice in the treatment of lesions of this character. Yet many bronchial adenomas cause remarkably little trouble for years apart from that attributable to partial or complete bronchial occlusion. On the contrary, fibrosarcoma is a much more malignant tumor and the results of inadequate therapy so much more disastrous than is the case with the usual bronchial adenoma that it does not seem that the two lesions can be fairly compared as to therapeutic requirements. We agree with those who feel that bronchial adenomas should be treated by pulmonary resection but in the case of fibrosarcoma of the bronchus, whose malignant nature is doubted by no one, such therapy appears to be a matter of much greater urgency. I do not believe that these truly malignant tumors can be reasonably managed in any other manner.

There appear to be adequate grounds for divergent opinions as to the extent of pulmonary resection indicated for bronchial sarcoma. It might well be argued that in dealing with this lesion, as with malignant disease in general, only a truly radical operation, in this instance a total pneumonectomy with thorough dissection of the mediastinal nodes, would suffice. However, despite the evidence that these lesions, as a rule, behave in a definitely malignant manner, there are at least some examples in which a simple lobectomy in the early cases can be

when a fibrosarcoma of bronchial origin is of such size and location that direct infiltration of the mediastinum may be reasonably suspected, or when, at the time of operation, the mediastinal lymph nodes are believed to be involved as a result of being found enlarged and of abnormal firmness. The finding of contained sarcoma cells in such a node following excision and examination by the frozen section technique would confirm the clinical impression and cause the surgeon to complete a thorough radical operation. However, even if one or more such nodes should prove to be negative, in the presence of the findings mentioned, a radical resection would probably be the wisest and safest course to follow.

SUMMARY AND CONCLUSIONS

1 Primary fibrosarcoma of the lung is a rare disease and lesions of this type arising in a bronchus have been reported in only a few instances.

2 A review of the literature indicates that primary bronchial fibrosarcoma has been diagnosed by bronchoscopic biopsy in only seven previously reported cases.

3 The small number of these lesions which have been encountered makes impossible the formulation of rational plan of therapy on the basis of experience.

4 In view of the malignant nature of this disease and the utter impossibility of determining by bronchoscopic observation the extent of its penetration into the lung, pulmonary resection is surely the therapy of choice. The known tendency of fibrosarcomas to be relatively radioresistant strengthens this opinion. Even though an occasional tumor of this type might prove to be partially radiosensitive, present concepts in the management of malignant pulmonary disease do not make acceptable the leaving of resectable sarcomatous tissue in situ and dependence upon irradiation for its control.

5 Three cases of fibrosarcoma of the bronchus which were diagnosed by bronchoscopic biopsy and treated by pulmonary resection are reported. Two of the three cases are considered definitely to have arisen primarily in a bronchus, the lesion in the third case involved a major bronchus, as proved by bronchial biopsy, but at the time of pneumonectomy the exact site of origin of the huge tumor could not be determined with certainty. Although not susceptible to proof because of the advanced state of the lesion at the time of resection, a primary bronchial origin of the lesion in that case is considered most probable.

6 Of the three cases reported herein, one patient was seen late in the course of his disease after his tumor, relatively small when first discovered, had been "watched" elsewhere until it had become huge. The extensive pulmonary invasion at the time of operation made total pneumonectomy mandatory. Palliation lasted for only a few months. Although not proved histologically, there was conclusive clinical evidence of early recurrence and distant metastases. Death occurred about nine months after resection. The second patient sought medical attention late and she also was treated by pneumonectomy although, in retrospect, it is felt that a right middle- and lower-lobe lobectomy might have been a satisfactory procedure. Palliation in that case was achieved for fifteen

months when signs of multiple distant metastases became evident. Death from metastatic fibrosarcoma occurred eighteen months after pneumonectomy. The third patient with a primary bronchial fibrosarcoma was seen early when her lesion was small and apparently localized. Resection was limited to removal of the right middle and lower lobes. She is alive and appears to be well thirteen months after operation.

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MERCURY-BAG BOLUS TO EXPEDITE PASSAGE OF ESOPHAGEAL DILATOR GUIDE STRING

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GRADUATED dilators guided within the lumen of the esophagus by passage over a slightly tensed, previously swallowed string were introduced by Mixer¹ in 1909, and are now in common use for the treatment of esophageal strictures due to a variety of causes. Usually a bead or split lead shot has been utilized as a weight and bolus to pull the swallowed string caudad until the traversed loops of intestine offer sufficient resistance to anchor the string. While the shot or bead employed may be small enough to pass through the narrowed zone and down the gastrointestinal tract carrying the string, the passage at both the area of stenosis and within the gastrointestinal tract may be erratic and time consuming.

Borrowing from the experiences of others in the rapid passage of intestinal decompression tubes,²⁻⁴ the substitution of a small mercury laden rubber bag, or gravity director head, as the bolus for the esophageal dilator guide string has assured its passage more quickly and more consistently. We have used the mercury bag bolus to expedite string swallowing only twelve times, but have been sufficiently impressed by the speed, safety, and reliability of this method to suggest its trial by others. Often the mercury bag propelled string can be swallowed at night and esophageal dilatation accomplished the following morning. Occasionally the dilator string has been passed expectantly in this simple manner and used for active dilatation if a stricture was found at esophagoscopy or abandoned if no stricture was encountered for the string in no wise interferes with esophagoscopy. By the weight of the mercury bag one is immediately aware of its passage through the stenotic area and after proper positioning of the patient its passage through the pylorus is frequently heralded, within a few minutes by a sudden movement of the string. This eliminates the necessity of observation periods or roentgenographic determinations.

The mercury bag bolus is used only as a means of carrying the string down the gastrointestinal tract and is not intended to serve as a bougie. The bag is not effective when it contains a small amount of mercury and is thus similar in its application to the head of theantor-intestinal decompression tube which has had failure of the mobility, liability, and great cohesion or unboundness of the causes of mercury. A convenient and reliable size for the bag is an ordinary rubber bladder. Two other alternatives of metallic mercury are

to permit free motion of the mercury, but not enough air to form a buoyant balloon from the absorption of intestinal gases⁵ The increased tension within the balloon could cause obstructive symptoms

Twelve feet of No 2 braided silk suture is used routinely as the swallowing string Usually there is adequate anchorage to tense the string after six to eight feet of it have been swallowed In one instance, when dilatation was delayed for three days, the mercury bag presented at the anus in forty-eight

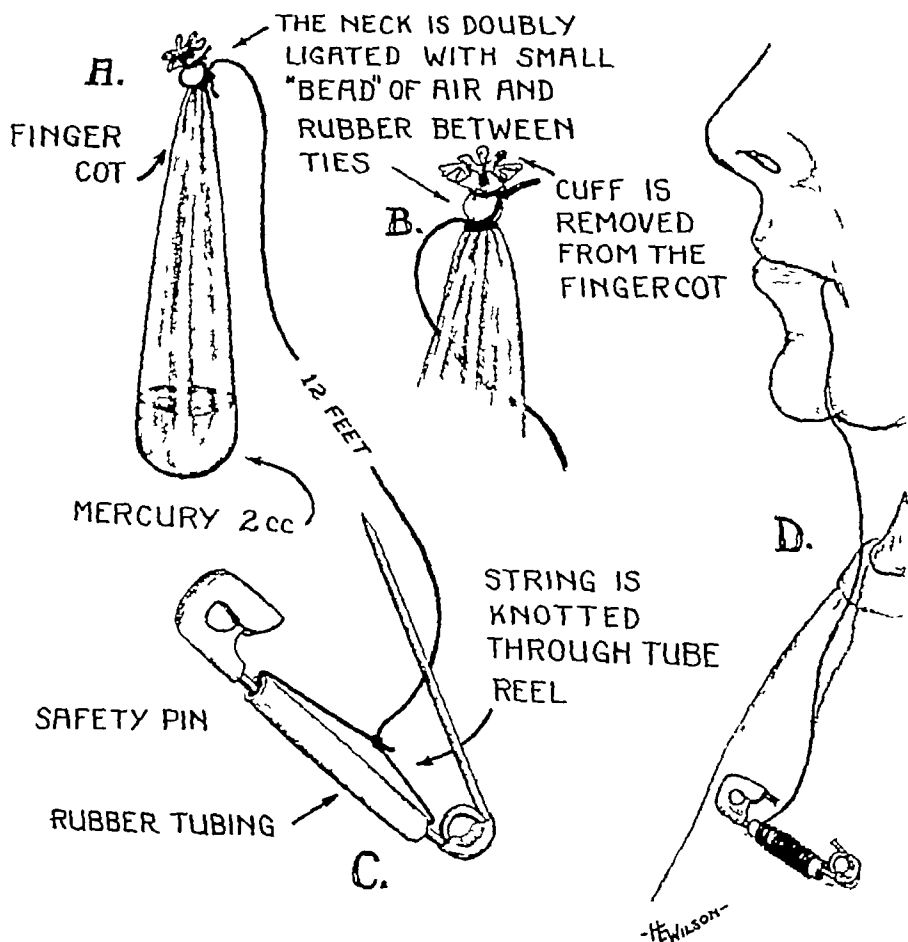


Fig 1--A Rubber finger cot with mercury B detail of method of bag closure which has proved leakproof and secure C rubber tube reel on keeper shaft of safety pin, stretch to place D mercury bag may be passed through the mouth as here, or via the nose and the string recovered from the mouth at the time of dilatation

hours and was cut free The patient suffered no ill effects from this pleating of his intestinal tract, and the retained string was satisfactory for esophageal dilatation the following morning

The mercury bag may be passed either through the nasopharynx or directly into the oropharynx as the patient prefers The use of a water-soluble lubricant

is especially advocated in patients with esophageal stenosis and should be mandatory if a topical anesthetic has been employed. When the bag is felt to have dropped into the stomach the patient is placed on his right side, rotated slightly forward in order to allow the bag to gravitate to the pylorus. Usually no further special positioning is necessary once passage through this sphincter is accomplished.

Although a string travels more rapidly down the intestinal tract when drawn along by a mercury bag than by other means nevertheless it is still important that the string not be swallowed at a more rapid rate than it is propelled by intestinal peristalsis. If swallowed at a greater rate than its passage at a lower level the string tends to snarl and form knots which later may obstruct passage of the bougie it is to guide. In order to accomplish this individualized rate of swallowing the string is wrapped, without twisting around a short piece of rubber tubing which has previously been threaded over the keeper shaft of a safety pin. When the mercury bag bolus is in the small intestine this improvised reel is pinned to the patient's clothing, the patient instructed to loosen only a short length of string at a time and cautioned to do this only when he is aware of traction from the swallowed string.

After dilatation of the esophagus is completed or abandoned the dilator guide string is cut low in the pharynx and the mercury bag with the remaining attached string allowed to pass per rectum. No difficulties have been encountered with spontaneous expulsion of the mercury bag bolus and attached string.

The use of the swallowed string for esophageal dilatation is contraindicated in the presence of an anatomic loop (such as gastroenterostomy or enteroenterostomy, etc.) at any level in the alimentary tract for the string may circle the loop and cut or ulcerate the mucosa as reported by Hubbard and Leven.

A mercury-bag bolus which is too large or too heavy may fail to pass the pylorus or, having passed, may pull so vigorously on the attached string that cutting of the esophagogastric or intestinal mucosa results. In the case reported by O'Leary and Burnett¹ a string used for retrograde dilatation was drawn into the jejunum by a displaced broken gastrostomy flange and peristaltic propulsion of the flange created a sawlike action of the string against the jejunum with resulting lacerations and perforations. A similar complication from excessive traction by the mercury bag bolus must be considered a possibility.

tube had ruptured. Lindemuth⁹ reported a fecal fistula which he considered to have been kept open by metallic mercury acting as a foreign body with granulomatous reaction. The fistula was at the drum site of appendectomy with perforation and peritonitis and the mercury had been spilled from the ruptured bag of an intestinal decompression tube. Yet Hoffman¹⁰ reports the accidental swallowing of mercury by a child with temporary trapping in the appendix and later spontaneous evacuation without harm. And Cantor¹¹ reports on fecal fistulas which had excreted mercury lost from an improperly tied bag on an intestinal decompression tube. The fistulas in this patient closed when a defunctionizing colostomy was done, because no mercury was permanently trapped within the fistulous tracts to act there as a foreign body.

SUMMARY

The use of a mercury-bag bolus to expedite gastrointestinal passage of the swallowed esophageal dilator guide string is discussed. The contraindications and potential complications of the technique are suggested.

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ANTERIOR MEDIASTINAL TRACHEOTOMY

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It is inevitable that an aggressive surgical approach to mediastinal and lower cervical disease should lead to an attempt to establish an airway elsewhere than in the cervical trachea. Methods for doing this have been discussed for about thirty years. Few actual cases, however, have been reported. Shoen and Fowler have recently described the successful establishment of a posterior mediastinal tracheotomy for the treatment of tracheal compression by a large aneurysm.¹ We should like to add to their report a case of aneurysm, involving the cervical trachea, treated by the cervical tracheotomy, treated by the establishment of an anterior mediastinal tracheotomy.

CASE REPORT

A five-year-old white male was first seen by a physician in March, 1919, at which time a laryngeal copy was done and a vocal cord biopsy obtained. This was reported to be a squamous cell carcinoma, and laryngectomy was advised. The operation was returned by the patient and in April, 1919, a tracheotomy was established followed by treatment of the patient from the present until Feb. 9, 1920, when he applied for admission to the hospital for terminal care. On admission he was found to be surrounded by a mass of inflammatory reaction. Intensive antibiotic therapy was initiated and on March 19, 1920, a partial tracheal abscess was incised and drained. This resulted in considerable improvement in the inflammation and much relief of symptoms. After the acute inflammation subsided the patient's condition was so favorable, and it was decided that surgical extirpation of the larynx was the only procedure which could offer even a remote chance of palliation. The rate of effort was explained to the patient and he consented to the proposed operation.

diameter and was located at the upper end of the tracheotomy partially occluding it, so that a tracheotomy tube was necessary to maintain the airway (Fig. 1). After consultation with the radiologist, it was decided that radiation therapy was feasible if the metallic tracheotomy tube could, in some manner, be removed from the tumor site. It appeared that the best way to accomplish this was to make a new opening, inferiorly and low in the trachea. In anticipation of this procedure a 14 mm polyethylene tube was obtained and molded to a suitable shape over a flame (Fig. 2).

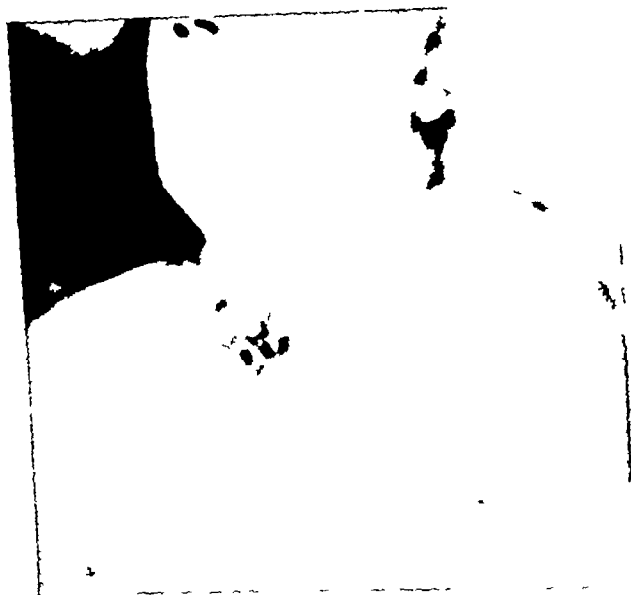


Fig. 1—Appearance of patient on admission. The large, recurrent carcinoma obstructs the tracheostomy to a degree which makes the constant wearing of the tracheotomy tube mandatory. Under these circumstances radiation therapy was felt to be impossible.

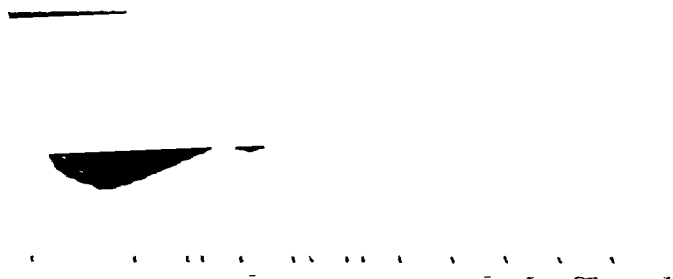


Fig. 2—The type of polythene tubing used for the tracheotomy. The angle was created by manipulating the tube over a flame.

On July 27, 1951, the manubrium of the sternum was resected under intravenous pentothal and ether anesthesia. The trachea was opened longitudinally in the anterior midline, and the polyethylene tube was inserted just above the innominate artery. The skin edges were partially approximated (Fig. 3). Good aeration was obtained, but a change of the tube every other day was necessary because of the tendency for mucous plugs to obstruct its lumen. Several additional polyethylene tubes were prepared for this purpose so that a sterile tube was always available. After a period of adjustment the patient was able to change these tubes himself, to aspirate the trachea and, in general, care for the new tracheotomy as well as the old.

Intensive deep x-ray therapy was now given which resulted in a remarkable melting away of the recurrent tumor (Fig. 4). During this period the tumor degenerated into a necrotic slough which produced a considerable amount of foul-smelling drainage, much of which ran down into the trachea. Aspiration of the necrotic material would have been an insoluble nursing problem had not the patient been able to spit out himself so easily. On Sept. 10, 1951, a razor graft was taken from the thigh and placed into the mediastinal defect to hasten epithelialization. The slough finally dropped, but an esophageal



Fig. 4—View from above of the patient shortly after removal of the necrotic material. This picture was taken just prior to the start of the radiation therapy.



stricture developed which required a Witzel gastrostomy (Oct 5, 1951) to maintain nutrition. Four months after the anterior mediastinal tracheotomy was established the opening into the anterior mediastinum was largely epithelialized, and the patient handled the new tracheotomy as readily as he did the old (Fig 5). On Nov 25 1951, his unhappy life was terminated by a sudden gush of blood from the mediastinal tracheotomy. An autopsy was done, and it was discovered that the esophageal obstruction was due to involvement by recurrent tumor rather than radiation fibrosis. There also was a lymphangitic extension of tumor deep into the mediastinum and into the upper one fourth of both lungs, as well as to the kidneys. Death was due to a linear perforation, about 5 mm long, in the posterior wall of the innominate artery. Although polyethylene tubing is extremely light, an agent other than its pressure could not be postulated as the cause of the perforation.



Fig 5—Appearance of the anterior mediastinal tracheotomy with the gauze packing removed. Note that the defect is practically completely epithelialized as a result of the razor graft.

DISCUSSION

The original plan of attack in this problem was to construct an anterior mediastinal tracheotomy, such as was done, and to line the defect with full thickness, pedicled skin grafts elevated from the chest, lateral to the tracheotomy, and rotated inward so as to form an epithelium lined tract. This procedure would have formed a skin-lined permanent opening and would have protected the adjacent innominate artery. Unfortunately, our hand was forced by the extremely vigorous growth of the recurrence which could not be controlled by local cautery applications. It was not considered wise to attempt complicated plastic procedures in the face of imminent intensive radiation therapy and the procedure was, therefore, delayed until after the radiation effect in the skin had subsided. If such a skin-lined tube can be constructed, or an anterior mediastinal tracheotomy is planned for temporary use only, several important advantages over the posterior mediastinal tracheotomy are apparent. First, and most important, it is readily accessible to the patient who

is able to care for it himself with only the usual training. Second, it is not in the way, so that the patient can wear his usual clothing and can rest upon his back when he sleeps. Although this case had an unfortunate end result, it was due to difficulties imposed by his disease. The operation itself holds some hope for the establishment of an airway in cases where a cervical tracheotomy cannot be made.

SUMMARY

A case in which an anterior mediastinal tracheotomy was prepared is reported.

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CARCINOMA OF THE ESOPHAGUS COMPLICATING CARDIOSPASM

REPORT OF A CASE

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THE subject of cardiospasm or achalasia of the esophagus has received considerable attention in the surgical literature, but emphasis has been focused upon the various technical procedures recommended for its relief. Although the differential diagnosis between cardiospasm and carcinoma of the esophagus has been adequately stressed, there is a prevalent impression that once the diagnosis of the former has been established, the possibility of the latter need not be considered. In an article entitled "Surgical Considerations of Achalasia" published in 1940, Ochsner and DeBakey¹ gave a comprehensive review of the world literature with 121 references, and the possible coincidence of carcinoma and cardiospasm was not suggested. A very recent clinical article² described an experience with 1,800 cases of cardiospasm, there was no mention of malignancy as a complication.

Evidently, the combination of achalasia and carcinoma is rare. However, there are a number of well-authenticated cases, mostly reported in the radiologic literature. In 1933, Vinson and Kennedy³ reported a case in which carcinoma developed seven years after the first hydrostatic dilatation. Kornblum and Fisher⁴ reported two cases in 1940. One of these had carcinoma when first seen, and the other developed it ten years after symptomatic treatment by hydrostatic dilatation. In 1944, Bersack⁵ presented the case of a 47-year old man with a history of dysphagia for ten years, with a large fungating carcinoma of the mid-esophagus. In 1947, Baei and Sieher⁶ were able to find only eight cases, including three of those mentioned above, which they would accept as genuine cases of carcinoma complicating achalasia, and added a case of their own. Because of the lack of clinical information, they did not include in their tabulation the three cases mentioned by Rake⁷ in his early brief communication. Within a brief interval an additional example came under Sieher's⁶ observation. It is of interest that only four of all the reported cases have been from the United States. Because of the rarity of the condition and some lessons which may be learned from the clinical history which terminated in the patient's death, the following case report is presented.

CASE REPORT

O B, a white man, aged 42 years, was admitted to the emergency room of the Henry Ford Hospital on Oct 30, 1950, with the complaint of vomiting bright red blood. Hematemesis of 5 or 6 cupfuls had occurred the day before and had been preceded by a tarry stool. He was dizzy and weak. A similar episode had occurred three years before. Considerable alcohol had been consumed for several years. The past history revealed

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Figs. 1-4. Anteroposterior roentgenogram of chest following the ingestion of barium showing markedly dilated esophagus lying principally in the right hemithorax. *A*, 1st roentogram taken at the same time. The irregularity of the anterior wall is probably significant.



Figs. 3-4. Anteroposterior roentgenogram of chest following the ingestion of barium showing markedly dilated esophagus lying principally in the right hemithorax.

gastrointestinal symptoms for more than ten years. These consisted of episodes of "vomiting," immediately or an hour after meals. The vomitus was never sour, hence it was assumed that the food had not reached the stomach. A radiologic examination in 1941 had demonstrated a greatly enlarged esophagus, but no treatment had been suggested. The provisional diagnoses were: bleeding peptic ulcer, bleeding esophageal varices, and cardiospasm with esophageal ulcer.

With cessation of bleeding, roentgen examination of the esophagus was carried out, and the following report was made: "Preliminary fluoroscopy of the chest revealed a large dense shadow extending half way into the right lung field and obscuring the right border of the cardiac shadow. As the patient was given barium, the shadow was realized to be the esophagus containing food and barium. The esophago-cardiac junction was poorly seen fluoroscopically and was visualized in only one of the films. There was much conical narrowing at the distal end of the esophagus with a markedly dilated, tortuous esophagus extending upward in the right chest to the level of the first rib. The stomach was partially filled. Impression marked cardiospasm. The possibility of an ulcer at the distal end of the esophagus should be considered." (Fig. 1)



Fig. 3—Autopsy specimen showing bifurcation of the trachea with probes passed through the two bronchoesophageal fistulas. There is bullous emphysema of the right pulmonary apex.

There was rapid improvement on an ulcer dietary regime, and an immediate operation for cure of the cardiospasm was recommended. This advice was not accepted, and the patient left the hospital. He returned for one outpatient visit on Nov. 25, 1950, at which time he had gained in weight and strength. Chest distress was absent, and there had been no evidence of bleeding. Nevertheless, he was again urged to have radical surgical treatment for the achalasia.



He was next seen on May 1, 1971, at which time he complained of vomiting, dysphagia, and indicated his willingness to undergo an operation. He was admitted on May 27 but by this time there had been a weight loss of 10 pounds and the vomitus contained blood. Roentgenograms showed some changes in the shape of the esophagus (Fig. 2). At this time, he developed a persistent cough which was productive of grayish yellow mucoid sputum. Tentatively, the pulmonary symptoms were believed to be the result of aspiration but to settle this point bronchoscopy was carried out on June 15. Intubation to see if there was seen posteriorly in both main bronchi and its removal was attended by lessened respiratory difficulty. Microscopically, the tissue removed from each of the bronchi proved to be squamous cell carcinoma (Grade 2). It was evident at this time that we were dealing with inoperable cancer and no effort was made to determine whether the lesion was primary in the esophagus or bronchus although the in-

stained mucopurulent material, the mucosal lining was thickened and hyperemic. A short distance distal to the crura on either side an irregular 3 mm orifice was found opening on the posterior wall of each main bronchus (Fig. 3). These communicated directly with a bulky ulcerated neoplasm occupying the anterior wall of the midportion of the esophagus. The thoracic segment of the latter save for the terminal 2 cm was enormously dilated to a circumference of 15 centimeters. A large irregular ulcerated tumor with elevated rolled edges occupied the segment immediately adjacent to the tracheal bifurcation (Fig. 4). The tumor except for the sloughing superficial portion was quite hard. Small vessels were apparent opening upon the irregular ulcer base and these were considered the source of the hemorrhage which filled the stomach. A smaller satellite lesion of similar character lay 2 cm distal to the primary growth. The remainder of the esophageal mucosa presented scattered patchy areas of thickening, roughening, and whitish discoloration regarded as leukoplakia. Inspection of the site of the cardiomyotomy showed that the constriction had been opened satisfactorily.



Fig. 5—Photomicrograph showing the transition from normal esophageal mucosa (right) to carcinoma (left)

Microscopically, the lesion in the esophagus consisted of an ulcerated mass of wildly proliferating anaplastic epithelial cells (Fig. 5). The cells grow coherently, forming irregular anastomosing cords which penetrated the wall of the esophagus. The cells, despite their anaplasia, were distinctly squamous in character, and numerous pearls with keratinized cores were observed. The fistulous tracts leading to the bronchi were lined entirely by carcinomatous tissue, and tumor undergrew and elevated the respiratory mucosa surrounding each of the bronchial communications. Corresponding to the leukoplakia observed grossly, there were patchy areas of cornification of the mucosa.

Additional pathologic findings were aspiration pneumonia, bullous emphysema of the right apex, and postoperative pleuritis of the left lower lobe.

DISCUSSION

It is obvious from a review of the case history that the presence of the complicating malignant lesion should have been strongly suspected when the patient was first seen with his second bout of hematemesis. Probably esophagoscopy

would have revealed the lesion. Assuming that carcinoma had been found in the anterior wall of the dilated esophagus at that time, an original plan of surgical treatment would have had to have been devised. Undoubtedly the conventional operation through the left chest with resection of the cardia and lower two thirds of the esophagus and high esophagogastrostomy would have been technically impossible because of the marked dilatation of the esophagus, its dislocation into the right thorax and probable early attachment to the bifurcation of the trachea. Accessibility would have been difficult because of the arch of the aorta. One could have considered a two stage procedure, the first operation being a Heller cardiomyoplasty, the second, a thoracotomy on the right with local excision of the carcinomatous ulcer and closure of the esophagus in continuity. The diameter of the huge esophagus would certainly have permitted this without any danger of stricture.

It is worth emphasizing that the symptom of dysphagia, the cardinal sign of carcinoma of the otherwise normal esophagus, is valueless in the diagnosis of malignancy in achalasia. First of all, some degree of dysphagia is already present, and second, in all the reported cases including our own, the cancer originated in the dilated middle third and obstruction had not been produced. In this connection, it should be pointed out that a number of standard pathology textbooks¹¹ state that carcinoma of the esophagus is more likely to occur at points of "physiologic narrowing." Such a tendency is certainly not evident in this group of cases.

Finally, one is tempted to speculate on the possibility that the development of cancer in the esophagus with cardiospasm may shed some light on the pathogenesis of this and other malignant lesions. The factor of chronic irritation with inflammatory changes and leukoplakia is certainly present. Also, there was a history of consumption of alcohol by our patient. This has been suggested as a possible predisposing factor but statistically no relationship has been demonstrated.^{12, 13}

SUMMARY

A case of carcinoma of the esophagus complicating cardiospasm is added to the small number previously reported. The advisability of the more frequent use of the esophagoscope is reiterated.

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SOME ASPECTS OF BRONCHIECTASIS IN INFANTS

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SINCE surprisingly little has been reported to date concerning the surgical treatment of bronchiectasis in the infant, we are presenting two cases which illustrate different etiologic factors and stress the feasibility of early surgical intervention.

CASE REPORTS

CASE 1—Baby P. Q., aged 21½ months. This infant was first seen at the Buffalo Children's Hospital Aug. 17, 1950, when 21½ months of age. The presenting complaint was paroxysmal cough since one week of age. This symptom had become progressively more severe and was accompanied with the production of tenacious mucus. Occasional emesis and cyanosis were noted following paroxysms of coughing.

On physical examination the child was well developed and well nourished. Attacks of coughing and considerable mucus in the pharynx were noted. There was dullness on percussion over the right lower chest accompanied by distant breath sounds and rales. A mild normocytic hypochromic anemia was present. A tuberculin patch test in dilution of 1:1000 was negative and tryptic activity was demonstrated in the stool.

A chest film showed a definite triangular density at the right base with associated areas of bronchopneumonia. Repeat roentgenographic studies revealed this abnormal density to be constant.

Treatment with antibiotics resulted in clinical improvement and the infant was subsequently discharged. However readmission was necessary within two months for the symptoms recurred. In the interim signs of malnutrition and chronic illness had become evident. The previous density in the right chest on the roentgenogram remained unchanged (Fig. 1).

Bronchoscopy was performed and bronchograms were made. This contrast study revealed a definite acicular bronchiectasis in the right lower lobe (Fig. 2).

Pulmonary resection was found upon and the infant was transferred to the surgical division for preparation. In December, 1950, a right lower lobectomy was performed. The specimen exhibited diffuse acicular bronchiectasis with purulent bronchitis (Fig. 3).

After an uneventful postoperative course the infant was discharged on the 14th day. Roentgenographic examination of the chest at the time of discharge revealed the pulmonary field to be clear (Fig. 4).

This patient illustrates the observations made by Wilson¹ in 1924. He found that aeration of the lower lobes was often incomplete for as long as ten weeks after birth. In this infant the onset of pulmonary disease was established during the first week of life. It appears that the pathological bronchopulmonary changes occurred within two months. This rapid pathogenesis illustrates how quickly bronchiectasis may develop when superimposed on atelectasis.

Field² studied 272 cases of primary cell carcinoma of the lung and concluded that the incidence of this group of

bronchiectasis is not comparable to that of pulmonary carcinoma.

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CASE 2—Infant J. D. aged 18 months. This patient was admitted to the Buffalo Children's Hospital with a history of recent exposure to tuberculosis. The symptoms, signs and laboratory studies confirmed the diagnosis of tuberculous meningitis. Specific therapy was started.

Dullness to percussion and rales were present over the right lower chest at the time of admission. Roentgenographic examination of the chest revealed a right lower lobe pneumonia.

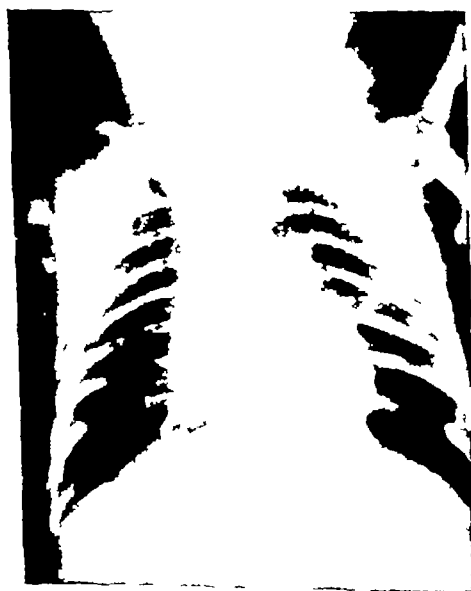


Fig. 1—Admission chest roentgenogram showing triangular shadow at right base.



Fig. 2—Bronchogram in oblique position. Note saccular dilatations of right lower lobe bronchi.

After intensive therapy, the infant improved, and was discharged from the hospital after a ten months' stay. It was noted upon discharge that the pneumonic process had failed to resolve. Atelectasis with bronchiectasis was thought to be present in this area (Fig 5).

In view of continuing clinical improvement, surgical resection of the involved portion of the lung was considered feasible. Roentgenographic studies, utilizing a contrast media, indicated bronchiectatic involvement of the right middle and lower lobes (Fig 6). Following adequate preparation a right middle and lower lobectomy was done.



Fig 3—Low-power microphotograph. Lumen of bronchi filled with exudate. Note bronchiectatic changes with evidence of chronic inflammatory reaction.



Fig 4—Chest roentgenogram taken at time of discharge from the hospital.

The pathologic specimen showed a diffuse cylindrical bronchiectasis. Atelectasis and interstitial infiltration with leukocytes were evident. Considerable fibrosis was noted surrounding the pulmonary arteries (Fig. 7).

No pulmonary disease was evident at the time of discharge (Fig. 8).



Fig. 5.—Chest plate taken at time of second admission. Note triangular hilar shadow on right.



Fig. 6.—Bronchogram in oblique projection. Note extensive saccular dilatations of right lower bronchi and suggestive involvement of middle segment.

A discussion of the preceding case must consider the abnormal pulmonary physiology resulting from a severe and protracted illness. The pathogenesis seems to be one of partial bronchial occlusion and atelectasis superimposed on an infectious pneumonic process.

Nelson³ is of the opinion that the bronchial obstruction and bronchial infection are basic mechanisms in the pathogenesis of bronchiectasis.



Fig. 7—Low power microphotograph. Note bronchial wall replaced with fibrous tissue. Evidence of chronic inflammatory changes and atelectasis.



Fig. 8—Chest roentgen gram taken at time of discharge from the hospital.

Pneumonia, along with other respiratory and exanthematous diseases, has been stressed as an important etiologic factor in the development of bronchiectasis. Field⁴ studied 160 cases of bronchiectasis in children as to possible predisposing conditions. Pneumonia and other respiratory infections were listed as predisposing factors in 70 per cent of her cases. Although tuberculosis and syphilis are to be considered, these diseases are probably responsible for only a very small number of cases.

REVIEW OF THE LITERATURE

Bronchiectasis has been estimated to constitute about 5 per cent of all childhood diseases.² Sauerbruch⁶ believed that in 80 per cent of the cases bronchiectasis could be traced to the early days of life. Bohler states that 50 per cent of adult patients date the onset of their disease to childhood. Perry and King⁵ reviewed 400 cases of bronchiectasis. Their findings indicated that in 69 per cent of the series, bronchiectasis occurred before the twentieth year of life, and 42 per cent occurred before the tenth year of life.

The etiology of bronchiectasis remains controversial and the presentation of a clear pathogenesis is often difficult. This bronchial disease has been categorically divided into congenital and acquired types. It may be said that, although many theories have been advocated, the true congenital form of the disease is rare and does not have a predilection for the lower lobes.² Engel¹⁰ believed that a congenital weakness of the bronchial walls occasionally exists. This inherent weakness plus respiratory infection might produce the changes of bronchiectasis. Meyer's¹¹ study revealed bronchiectasis in siblings to be fourteen times more frequent than in the general population. Nelson's³ group believed that the true congenital form of bronchiectasis and cystic disease of the lung were closely allied.

Acquired bronchiectasis has many varied etiologic factors. Exanthematous diseases, pertussis, respiratory infections, foreign body aspiration, sinusitis, and pneumonia have all been suspected. Atelectasis with associated bronchial infection seems to be the immediate causative factors.

The mortality and morbidity rates of bronchiectasis are high. In Table I are tabulated mortality figures of untreated bronchiectasis.

TABLE I

SOURCE OF INFORMATION	NUMBER OF CASES	MORTALITY (PER CENT)
1 Perry and King ⁵	400	26
2 Selby ¹²	50	66
3 Roles and Todd ¹³	49	46.9
4 Bradshaw, Putnev & Clerf ¹⁴	171	34.5

*A twelve-year follow up study.

Lebert¹³ analyzed fifty-two autopsied cases of bronchiectasis. Of this group, 21 per cent had died within the first year of the disease, and 75 per cent were dead within ten years of the onset of the disease.

Certainly, the above figures indicate that bronchiectasis has a high mortality rate within a short period of time.

Some years ago Brennemann¹⁵ stated that the cure of bronchiectasis lay in the scope of early surgery. The argument would seem to be how safely can such procedures be carried out rather than whether surgery should or should not be done. The two cases we are reporting illustrate the feasibility of early operation in the infant. The case of Baby P. Q. appears to be the youngest age recorded in the literature for extirpation of bronchiectatic pulmonary tissue. Obviously, younger infants have been operated upon for congenital cystic pulmonary disease.

The surgical mortality rate in infants and children seems remarkably low as compared with the mortality rate of medically treated bronchiectasis.

Results in the surgical treatment of bronchiectasis in infants and children are shown in Table II.

TABLE II

SOURCE OF INFORMATION	NUMBER OF CASES OPERATED	MORTALITY (PER CENT)
1 Field ¹⁶	50	2
2 Strieder ¹⁷	25	0
3 Edwards ¹⁸	38	0
4 Nelson ³	25	0

Other authors (Riggens,¹⁹ Buckles,⁵ Field,¹⁶ Bohrer²¹) point out the morbidity and occupational loss suffered by these patients. The inevitable picture of advanced bronchiectasis is one of chronic invalidism, mental lethargy, and social maladjustment.

Bohrer and Lester²⁰ predicated that such cases will be referred for surgical treatment more frequently as pediatricians realize the comparative safety of operative intervention. While no one denies bronchiectasis can be treated medically, such treatment requires endless, intelligent, methodical attention.

SUMMARY

Two infants developing bronchiectasis while under observation are described. In both, bronchial obstruction with infection was the apparent cause. In one infant the obstruction developed in connection with an unexpanded lower lobe. In the other, the obstruction was associated with the depression accompanying a tuberculous meningitis. Both were completely relieved of their pulmonary disease by operative extirpation.

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THYMIC NEOPLASMS

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INVESTIGATION of the literature pertaining to thymic neoplasms reveals disagreement and confusion. Perhaps much of this is due to the rarity of the lesion. Crosby¹ in 1932 reviewed all the cases of malignant thymic tumors up to that time and found only 166 cases. There have been, subsequently, approximately 100 additional cases on record. However, until recently² there has been no single large series reviewed and recorded by one group of observers. It becomes apparent that many of the reviews of the subject offered little more than the perpetuation of controversial and diversified opinions and observations.

Another source substantiating the lack of agreement regarding these tumors has been the varied opinions about the embryogenesis of the thymus gland and the exact identification of its cellular components. Clarification is, of course, necessary before a proper classification and understanding of these neoplasms can be obtained. It is the general consensus that the so called thymocyte is, in truth, a lymphocyte, and that the so called reticulum cell, or reticuloepithelial cell, is epithelial in nature arising from the entodermal epithelium of the third branchial cleft. These are the cells which form Hassall's corpuscles. Reference to these as reticulum cells appears unwarranted as well as confusing, since it bears no relationship, embryologically, to the reticulum cells observed in the tissues of the reticuloendothelial system. It becomes apparent, then, that primary neoplasms of the thymus gland would be either lymphoid, epithelial, or of a type demonstrating a combination of these elements. Although in Crosby's review¹ two-thirds of the neoplasms were considered as lymphosarcomas, nevertheless reference is made to the occurrence of epithelial elements tending to form structures similar to Hassall's corpuscles as a means of identifying the thymic origin of such neoplasms. Other reported instances of lymphosarcoma similarly mentioned the presence of epithelial cells in their microscopic descriptions. The lymphoid tumor is emphasized by Willis³ and inferred by Scybold and associates² are exceedingly rare. Foot,⁴ as well as Symmers,⁵ also considers the mixed epithelial and lymphoid type the most common. With this rather simplified concept of thymic structure one is able to arrive at a rather practical classification. The following types are recognized: (1) lymphosarcoma, (2) carcinoma, (3) lymphoepithelioma (similar to the lymphoepitheliomas encountered in the upper respiratory tract), and (4) carcinoma, granulomatous type. The latter group has been well described by Fowenhiupt⁶ as representing tumors of immature thymic epithelial cells intermingled with a polymorphous in-

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inflammatory infiltrate and a collagenous connective tissue stroma. The resultant picture is not unlike that of Hodgkin's granuloma, yet, if many areas of the tumor are studied, transitions to the more mature epithelial cell type and more familiar lymphoepithelioma pattern are recognized. This granulomatous variant is also recognized by Seybold and associates² as being epithelial in origin. Probably many of the reported instances of primary Hodgkin's disease of the thymus gland represent this dysmaturity in thymic carcinoma; another variant described by Symmers, Helwig, and Foot is the perithelial type. However, since many of the neoplasms described in the other groups often demonstrate a perithelial pattern, the addition of this type to a classification of thymic neoplasms seems unnecessary. Although the rather non-committal term of thymoma, first used by Grandhomme³ in 1900, has been employed by many authors, confusion has been created because some investigators have chosen the term as referring to those neoplasms only of lymphoid structure, while others have used it with reference to the epithelial neoplasms of the thymus gland. With a more unified opinion as to the embryology of the thymus gland and an understanding of its cellular components, the term might well be discarded to avoid confusion and redundancy.

The segregation of tumors arising in mediastinal lymph node and adjacent pulmonary parenchyma may offer difficulty in the exact identification of some thymic tumors, especially since thymic neoplasms tend to invade contiguous structures by direct invasion. However, the vast majority of thymic neoplasms should be recognized, after careful histologic and roentgen study and gross anatomic investigation.

The association of thymic abnormalities with myasthenia gravis has also added a certain element of confusion to the subject of thymic tumors. One obtains the impression, from reports of Bell,⁴ Lacey,⁵ and Homburger,⁶ that a distinct type of neoplasm is associated with myasthenia gravis. These investigators were of the opinion that the tumors were epithelial in type. However there are those^{7, 8} who have contended that they are of a lymphoid nature and Norris⁹ has stated that these instances of thymic abnormality associated with myasthenia gravis represent a hyperplasia. Seybold and co-workers² have, however, more recently demonstrated the similarity between the neoplasms associated with myasthenia and those tumors unaccompanied by this disease.

We shall report five instances of thymic neoplasms which tend to support many of the newer concepts and emphasize certain important clinical features of thymic tumors.

CASE 1. A 27-year-old married white woman was admitted to the Cleveland Clinic Hospital because of thyroid enlargement. The patient had experienced weakness, anorexia, cough, and progressive weight loss for two months prior to admission.

Physical examination revealed a rather well developed, poorly nourished woman with pronounced pallor. Examination of the neck revealed the presence of a firm, nontender nodule overlying the superior pole of the right lobe of the thyroid. There was no cervical lymphadenopathy. There was limited expansion of the left side of the thoracic cage, and decreased breath sounds over the upper half of the left side of the chest posteriorly. A harsh systolic murmur was heard over the aortic area. The pulse was regular (72 per minute),

Blood pressure was 130/90. Abdominal examination revealed the presence of a large, firm, slightly tender mass in the right upper quadrant extending to the right flank. It did not appear to be continuous with the liver.

Laboratory examination revealed 3,900,000 red blood cells, hemoglobin 10.8 Gm, and a white blood count of 5,900, with normal differential. Urinalysis was negative, as were Kahn and Wassermann tests.

Roentgenograms (Fig 1, *A* and *B*) of the chest demonstrated a large, soft tissue shadow extending from the left hilum superiorly and anteriorly. The mass had a smooth border and was somewhat lobulated. The right lung showed several soft tissue nodules in both the upper and lower lung fields. The heart appeared to be normal. Urography demonstrated inferior displacement of the right kidney with incomplete filling of the superior calyx. The opposite kidney was normal in configuration.

Hospital Course—The thyroid nodule was removed for histologic examination.



Fig 1—*A* and *B* Anteroposterior and oblique roentgenograms of the chest, Case 1

Microscopic Examination—(Fig 2) Sections stained with hematoxylin, eosin, and methylene blue revealed a neoplasm replacing all but a few thyroid follicles. The neoplastic cells were polygonal, cuboidal, and round in shape with pink staining cytoplasm, many of which were indefinite. The cell nuclei were for the most part vesicular, with moderate irregularity. An occasional mitotic figure was noted. The tumor cells were arranged in clusters which, in areas, showed a tendency toward whorl formation. An occasional lymphocyte was present among and between cell masses.

A Wilder's reticulum stain demonstrated a fine reticulum about some of the cell groups. However, there was no reticulum manifested about or between individual cells.

Diagnosis—Carcinoma of thymic origin secondary in the thyroid. The patient was discharged from the hospital, and is currently receiving roentgen therapy on an outpatient basis.

CASE 2—A 45 year old white man entered the clinic with rather severe periodic cramping pain in the right flank. Sixteen years prior to this complaint, he had noticed difficulty in swallowing and some associated generalized numbness and weakness. This had progressed and on occasion had become severely incapacitating. A diagnosis of myasthenia gravis, after therapeutic tests, was made three years later. The patient stated that the use of prostigmine had given him exceptional relief. He remained well controlled with the use of several ampules

of prostigmine daily. About four years prior to his recent complaint he began to have bouts of postprandial diarrhea. With the onset of the diarrhea, the severity of the generalized weakness became minimized and his prostigmine requirement also decreased. The patient had lost 100 pounds during the last three years. At the time of admission he was taking one ampule of prostigmine which afforded relief from weakness for twenty four hours.

Physical examination revealed a tall, somewhat emaciated white man in no apparent acute distress. Examination of the head and neck was negative, as was examination of the chest. Several small, discrete, posterior cervical lymph nodes were palpable. Blood pressure was 110/60. The liver, kidney, and spleen were not palpable. Slight tenderness was elicited on deep pressure over the right flank.

Laboratory studies showed hemoglobin of 13.5 Gm, and white blood count 17,750. Urinalysis, Wassermann, and Kahn tests were negative.

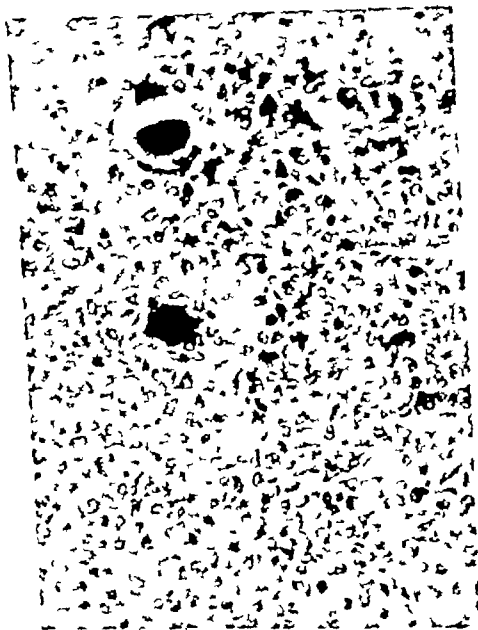


Fig 2—Thymic carcinoma in thyroid with few follicles remaining principally epithelial (X275)

Roentgenogram of the chest revealed tracheal compression at the left of D4 and D5, with slight deviation to the left. There was narrowing of the trachea just below the sternal clavicular junction with no obstruction. A soft tissue mass to the right of the trachea at the level of the clavicle was noted. The left lung field was clear. Moderate pleural reaction was apparent over the upper right lung field with a soft tissue infiltration at the base. A number of circular areas, with increased density suggesting pleural and parenchymal metastatic lesions, were present on the right. Examination of the skeletal system was negative. Cholecystography and gastrointestinal roentgenograms, as well as an intravenous urogram, were negative.

A thoracotomy was performed, at which time a large tumor mass was discovered extending from the anterior mediastinum to the adjacent right parietal pleura. Several tumor nodules were noted in the right lung and one was removed for histologic examination.

Microscopic Examination—(Fig 3, A) Sections stained with hematoxylin, eosin, and methylene blue revealed a neoplasm formed by masses of cuboidal, and polygonal shaped cells with indefinite pink staining cytoplasm. The cell nuclei were irregular and for the most

part vesicular with occasional hyperchromasia, mitotic figures and a rare atypical mitosis. The tumor contained moderately dense connective tissue trabeculae. In areas there was whorling of the epithelial cells resembling Hassell's corpuscles. The epithelial elements appeared to lie in a stroma composed of mature lymphocytes, some of which were intermingled with the epithelial components of the tumor. Approximately equal proportions of lymphoid and epithelial elements were present.

Sections treated with Wilder's reticulum stain revealed a moderately dense reticulum about cell groups.

Diagnosis—Lymphoepithelioma of thymic origin in lung. The patient was given a course of nitrogen mustard therapy and discharged from the hospital. To date, one and one half years after admission, his general condition has been satisfactory.

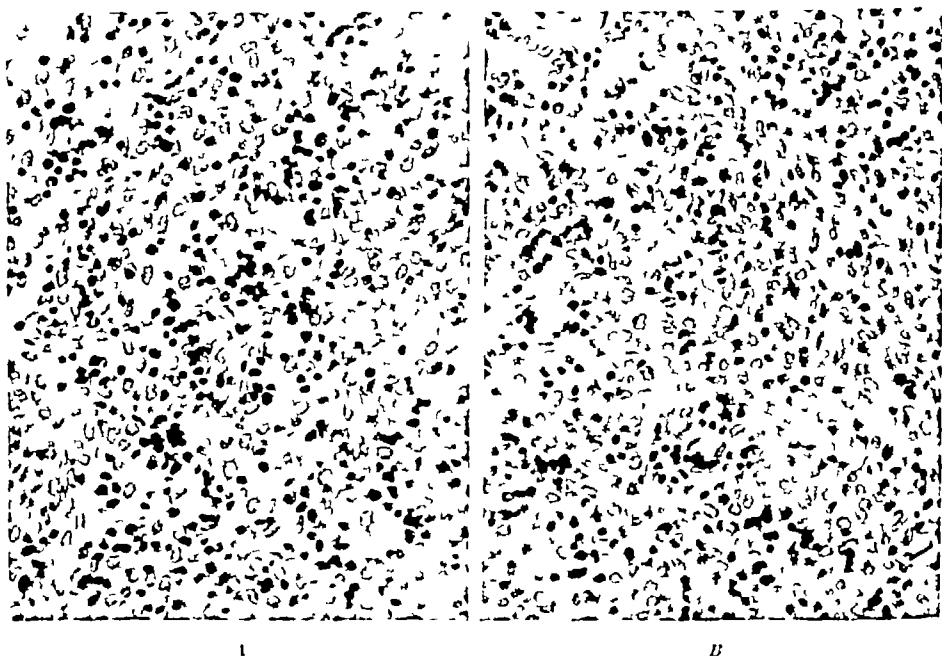


Fig. 3—1 Photomicrograph of neoplasm from Case 2. Patient had myasthenia gravis ($\times 275$). B Section of neoplasm from Case 4. Patient did not have myasthenia. Identical histologically to 3-1 ($\times 275$).

CASE 3—The patient was a 58 year old white man. For three months prior to admission he had noticed progressive weakness, slurring of speech, and difficulty in elevating his arms. He had also recently experienced difficulty in closing his jaws. All these symptoms were aggravated by any form of physical exertion, and appeared to become progressively worse throughout the day. An x-ray examination of his chest, taken two years prior to admission, had disclosed the presence of a mediastinal tumor.

Physical examination revealed a well developed, well nourished man with a thick, slurred speech which was barely intelligible. He showed considerable difficulty in elevating his arms. Examination of the head, neck, and chest was essentially negative. Blood pressure was 130/90. Examination of the abdomen was negative. The clinical diagnosis of myasthenia gravis was confirmed by the dramatic response to pyridostigmine.

Electrocardiographic studies were negative. Urinalysis was also negative. Hemoglobin was 14 Gm, and the white blood count was 7,500.

Roentgenogram of the chest demonstrated small, scattered calcifications in the left hilar region and left pulmonary parenchyma. There was a smooth, homogeneous mass in the right hilum and anterior mediastinum.

An anterior mediastinotomy was performed through a sternal splitting incision. An oval shaped neoplasm situated in the anterior mediastinum overlying the aorta was encountered. The tumor measured approximately 5 by 3 by 2 cm. The neoplasm apparently was encapsulated and was rather easily removed in toto.

Pathology—(Fig 4, A and B) The specimen consisted of an ovoid mass weighing 72 Gm. and measuring 4 by 3 by 2 cm. At one pole a conchlike prolongation was evident. The outer surface was covered by a smooth, glistening, transparent, whitish gray capsule. The cut surface revealed the main mass to be comprised of yellowish gray, smooth, glistening tissue with interspersed, irregular areas of yellowish gray necrosis. Section through the prolongation revealed yellowish gray tissue with irregular areas of hemorrhage.

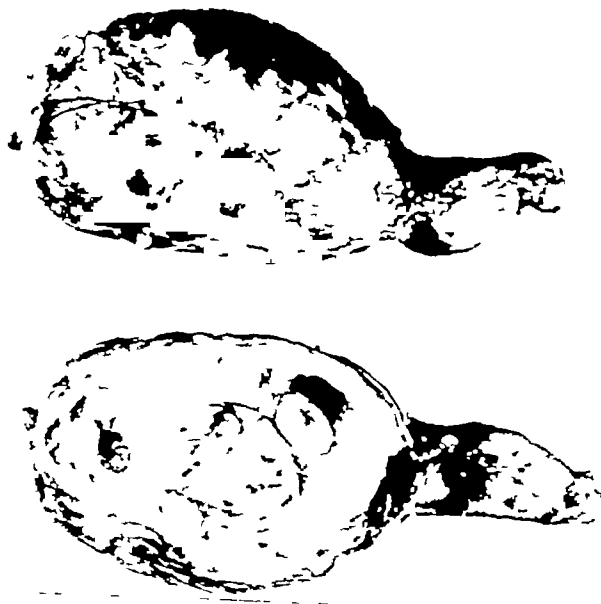


Fig. 4—A and B Outer and cut surfaces of neoplasm removed in Case 3

Microscopic Examination—Sections stained with hematoxylin, eosin, and methylene blue revealed a neoplasm comprised principally of mature lymphocytes. However, irregularly shaped, rather large, epithelial like cells with pink staining cytoplasm and irregular, somewhat vesicular nuclei were present among the lymphoid tissue arranged in small masses and clusters. An occasional mitotic figure was evident, as well as nuclear hyperchromasia. In areas a tendency to whorling was evident. Moderately dense bands of collagenous connective tissue trabeculae coursed throughout the tumor. A dense collagenous connective tissue capsule was apparent about all sections studied.

Diagnosis—Lymphoepithelioma of the thymus. The postoperative course was uneventful and the symptoms of myasthenia gravis improved so that the patient no longer required prostigmine medication. The patient was well for approximately two and one-half years. However, a recent report (Fig 5, A and B) has revealed that he has been conneed to another hospital because of recurrence of the anterior mediastinal tumor as well as reappear

ance of the symptoms of myasthenia gravis, which now require prostigmine therapy. There are also symptoms indicating superior vena caval obstruction. The patient is receiving external x-ray therapy to the lesion.

CASE 4—A 37 year old white woman was admitted to the Cleveland Clinic for a routine physical examination. At the time she had no complaints. A routine examination, four years previously, revealed a normal roentgenogram of the chest.

Physical examination was essentially negative.

Laboratory studies revealed a red blood count of 5,100,000 cells, white blood count 15,675 with a normal differential, hemoglobin 12.5 Gm. Urinalysis was negative, as were blood Wassermann and Kahn tests. Stool examination revealed the presence of *Endameba histolytica*.

Chest roentgenogram demonstrated a large, rounded shadow projecting outward from the right mediastinal area. The mass had a diameter of 8.5 cm, and projected at least 4 cm lateral to the right of the spine. Lateral films revealed that the mass was located in the anterior mediastinum. Roentgen examination of the gastrointestinal tract was negative. The patient refused thoracotomy at that time. However, several months later the patient was readmitted to the hospital because of shortness of breath and pain in the right side of the chest.



Fig 5—A and B Anteroposterior and oblique roentgenograms of recurrent neoplasm in Case 3

Physical examination at the time of the second admission revealed a vigorous, healthy looking, well developed, well nourished, white woman in no apparent distress. Examination was essentially negative, except for moderate decrease in expansion of the right side of the chest with decreased breath sounds at the right base and flatness to percussion in the same area. The remainder of the examination was negative.

Exploratory thoracotomy was performed under endotracheal anesthesia. An inoperable anterior mediastinal tumor with extensive pleural metastases was discovered. Pleural implants were rather large, varying from 3 to 6 cm in cross diameter. The neoplasm also had invaded the right lung by direct extension. A biopsy was taken from one of the pleural implants.

Microscopic Examination—(Figs 3, B and 6) Sections stained with hematoxylin, eosin and methylene blue revealed a neoplasm formed by two types of cells, each occurring in equal quantity. One cell type was mature lymphocytes, the other which demonstrated sheetlike and

cluster arrangements appeared epithelial. The epithelial cells were cuboidal and polygonal in shape although their cytoplasm was frequently indennite. Nuclei were, for the most part, vesicular and irregular in shape. Nuclear borders were well denned. Occasional mitotic figures were frequent as well as a rare atypical form. Occasional nuclear hyperchromasia was also present. The epithelial masses were intermingled with the lymphocytes noted. A tumor thrombus comprised of both epithelial and lymphoid elements was present in a large endothelial lined space. Wilder's reticulum stain failed to reveal any reticulum associated with the neoplasm.

Diagnosis—Lymphoepithelioma of thymic origin in pleura. Nitrogen mustard therapy was administered. The patient was discharged from the hospital three months ago.



Fig 6.—Tumor thrombus in endothelial lined space from neoplasm in Case 4 (X70)

*CASE 5**—A 37-year old white housewife presented herself for examination with the chief complaint of occasional epigastric distress and a sense of fullness in her neck. Roentgenogram of the chest one year prior to admission to the hospital was negative. There were no other complaints.

Physical check up revealed a well developed, thin, 37 year old white woman in no acute distress. Head, neck, and lungs were normal. Blood pressure was 104/76, pulse was 96 per minute and regular. The remainder of the examination was noncontributory.

Laboratory Examination—Erythrocyte count was 4.71 million, and hemoglobin 13.2 Gm. per cent, the white blood count was 8,400. Urinalysis was negative, as were Kahn and Wassermann tests.

Roentgenogram of the chest revealed a dense shadow in the anterior superior mediastinum.

A thoracotomy was performed, and a large portion of a lobulated, nonencapsulated mediastinal neoplasm was removed. It was noted that there was extreme extension into the upper portion of the left lung.

*This case was reviewed through the courtesy of Dr. Philip Graf and Dr. Earle B. Kay, Charity Hospital, Cleveland, Ohio.

Microscopic Examination—(Fig 7, *A*) Sections of neoplasm removed from the mediastinum revealed a granulomatous like lesion comprised of large, irregularly shaped epithelial like cells, measuring approximately 40 microns in diameter. Cytoplasm of these cells were for the most part indefinite and pink staining. Nuclei were large, in many instances lobate or horseshoe shaped with prominent nucleoli. These epithelial like cells lay individually scattered among a polymorphous infiltrate comprised of lymphocytes, occasional neutrophils, and rare eosinophil. There was also a moderately dense, relatively acellular, collagenous connective tissue stroma with tendency toward trabeculation. In some areas there was pronounced necrosis.

Sections (Fig 7, *B*) of neoplasm from the lung revealed masses of epithelial cells arranged in sheets and clusters somewhat similar to those noted in the mediastinal tumor. However, they were smaller and more uniform in appearance, with round nuclei. These epithelial masses lay in a stroma comprised principally of lymphocytes.

Diagnosis—Carcinoma of thymus, granulomatous type.

The patient's postoperative course was uneventful, and she is now receiving roentgen therapy.

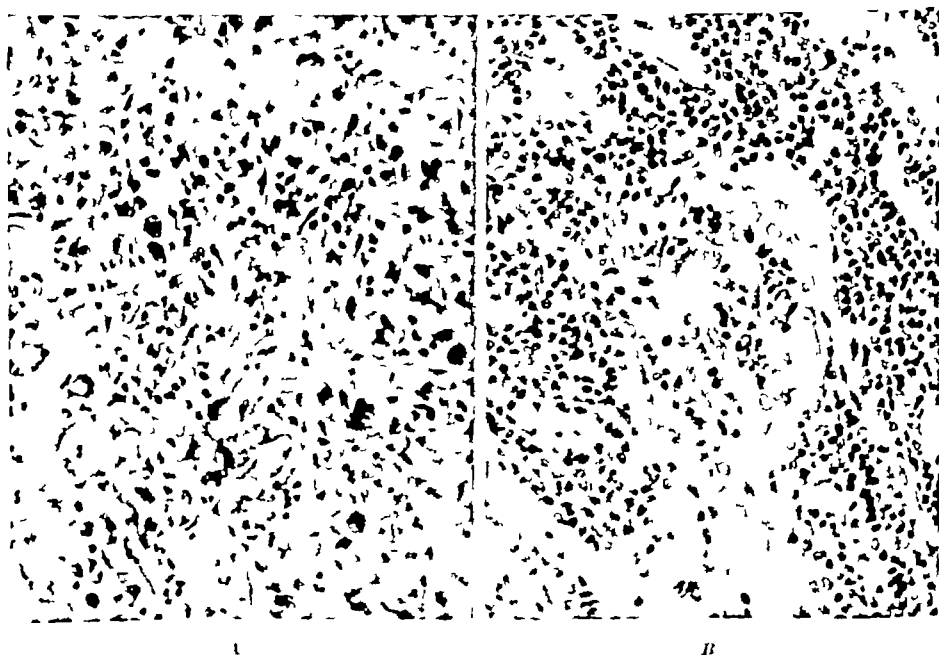


FIG. 7—*A* Section of neoplasm in Case 3 from main mediastinal mass. Note granulomatous appearance with large atypical cells ($\times 275$). *B* Section of neoplasm in lung from same case demonstrating more epithelial like cells ($\times 275$).

DISCUSSION

Although none of the cases presented have been autopsied nevertheless the histologic appearance of the neoplasms as well as their roentgenographic and gross features at the time of thoracotomy substantiates their thymic origin. The fulfillment of the identifying features of thymic neoplasms is stated by Jones.¹² These are: (1) large lobulated tumors at the site of the thymus, (2) extension

behind the sternum without infiltration of the bone, (3) involvement of the pericardium and pleura by direct extension, and (4) a resemblance to thymic tissue on histologic examination

Histologically the tumors are similar in that they contain both epithelial and lymphoid elements in varying proportions. The neoplasms from patients in Cases 2 and 4 demonstrated equal lymphoid and epithelial components, whereas the tumor in Case 3 was predominately lymphocytic and that in Case 1 predominately epithelial. The neoplasm classified as carcinoma granulomatous type resembled those tumors recently described by Towne¹⁰. Although they present a microscopic picture not unlike that of Hodgkin's disease their true epithelial nature has been well explained by indicating their resemblance to immature thymic epithelium. The interpretation of the histologic appearances of the five tumors studied resembles the more recent concepts of the structure of thymic neoplasms.

A perplexing question for the pathologist to answer is whether a given thymic neoplasm is benign or malignant. Certainly this distinction cannot be made from histologic study alone, as evident from the study of the neoplasms reported here or the much larger series of Seibold and associates.² All of the neoplasms studied demonstrated some nuclear hyperchromasia, irregularity and pleomorphism as well as an occasional pathologic mitosis in one. Further these tumors show a definite tendency toward invasion of contiguous structures, and the occurrence of distant metastases in a large proportion of cases as indicated by Crosby¹ and Willis³ makes it apparent that all thymic neoplasms must be regarded with extreme suspicion. Even with apparent encapsulation and complete removal as in our Case 3 recurrence occurred two years subsequent to surgery. This further emphasizes the necessity for caution in the prognosis of thymic tumors.

As has been previously mentioned, there are no histologic criteria which enable one to differentiate the thymic neoplasm associated with myasthenia gravis from those tumors unassociated with this disease. The concept that malignant thymic tumors are not associated with myasthenia gravis must be altered. DeRow and co-workers¹⁶ have stated that approximately 10 per cent of thymic neoplasms associated with myasthenia gravis encroach upon contiguous structures, and they have reported a case with intrapulmonary metastases. Our Case 2 is quite similar, in that pulmonary metastases were also present.

It is interesting to note that in this small group of thymic neoplasms only one presented symptoms indicating mediastinal obstruction, the classical symptoms associated with these tumors. In one, the first clinical manifestation was the presence of a metastatic nodule in the thyroid. One was discovered during a routine roentgen examination of the chest. This latter patient had numerous pleural implants at the time of thoracotomy, yet had no symptoms at the time the lesion was first discovered. Two of the tumors were associated with myasthenia gravis. It has been estimated that 75 per cent of the patients with thymic tumor have myasthenia gravis, whereas 50 per cent of the patients

with myasthenia gravis will also demonstrate an associated thymic tumor²

The cases of thymic tumor associated with myasthenia gravis are unusual from a clinical standpoint. One patient, Case 2, experienced definite relief from the symptoms of myasthenia gravis which had been present for thirteen years with the apparent onset of the thymic neoplasm, and his prostigmine requirements were reduced suggesting that a prostigmine-like substance was being elaborated by the neoplasm. This effect was also manifested by the occurrence of intestinal hypermotility. Microscopic study of the neoplasm offered no explanation for such behavior since it resembled other tumors in the group. Although pharmacologic studies of these tumors have been attempted, their results have been discouraging. No similar case of thymic neoplasm and myasthenia gravis has been reported in the literature. The other patient with the thymic tumor and myasthenia gravis experienced relief from his myasthenia following the removal of a thymic neoplasm. Exacerbation, however, occurred with recurrence of the tumor. These unusual features as well as those in the patient described by Fehrshtand and Shaw¹⁷ (in whom the symptoms of myasthenia developed after the removal of a thymic tumor) demonstrate not only the unpredictability of the clinical course in patients with myasthenia gravis and thymic tumors, but also our inadequate interpretation of their relationship.

The cases reported have not been followed for sufficient periods to allow for the evaluation of therapy. However, it might be stated that the use of nitrogen mustard therapy or x-ray therapy has been disappointing. This has been the experience of others who have reported on longer follow up periods on this type of treatment. It appears that, when possible, the treatment of choice is surgical excision of the neoplasm. Seybold and associates⁷ have observed no recurrences in their group treated in this manner.

SUMMARY

The literature pertaining to thymic neoplasms is reviewed, with an attempt toward presenting a simplified understanding of terminology, embryology and classification of these neoplasms.

Thymic tumors may be classified into (1) lymphosarcoma, (2) carcinoma, (3) lymphoepithelioma, and (4) carcinoma, granulomatous type.

Five cases of thymic tumor are reported. One of the patients with myasthenia gravis studied demonstrated pronounced diminution in the symptoms and prostigmine requirements of a preexisting myasthenia gravis with the apparent development of a thymic tumor, suggesting that a prostigmine-like substance was elaborated by the neoplasm.

The frequent occurrence of invasion of adjacent structures, as well as the occurrence of distant metastases and lack of histologic difference between so called benign and malignant thymic neoplasms, indicates that all such tumors must be regarded with suspicion. The recurrence of a tumor which had been apparently encapsulated and completely removed two years after operation (Case 3) emphasizes the caution necessary in prognosticating the clinical behavior of these neoplasms.

There is no histologic criteria by which the thymic tumors associated with myasthenia gravis can be differentiated from those unaccompanied by this disease

Thymic tumors unassociated with myasthenia gravis may be asymptomatic. Only one of the three patients without myasthenia gravis presented the more classical symptoms of thymic tumor.

It appears that surgical extirpation is the treatment of choice although many cases on initial observation are inoperable. Nitrogen mustard therapy, or x-ray therapy, has been for the most part disappointing in the inoperable cases.

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INDICATIONS FOR A DELAYED ROENTGENOGRAM IN BRONCHOGRAPHY

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IN MAPPING out the extent of surgical resection for a patient with bronchiectasis, a delayed film following bronchography may prove to be of considerable value. Often important in establishing the presence of bronchiectasis, such a delayed film may also help avoid needless sacrifice of tissue by eliminating uncertainty concerning a specific bronchial segment. Lung tissue may thus be conserved without the risk of compromising a complete cure.

Three cases have been selected to demonstrate the value of a delayed roentgenogram in bronchography.

CASE 1—Bronchography was performed for preoperative localization of bronchiectasis. Definite dilatation of bronchi was disclosed in the right middle lobe (Fig 1, A). There also appeared to be obstruction and some cylindrical enlargement of an adjacent bronchus in the anterior segment of the right upper lobe (arrow). In the delayed film (Fig 1, B) obstruction to the latter bronchus is seen to have been overcome. Return of the bronchial caliber to normal with filling of its peripheral branches excluded any need for removal of this lung segment (arrow).

CASE 2—Bronchography was performed in this patient to determine the status of the bronchial tree. Extensive sacular and cylindrical changes were demonstrated in the left upper lobe bronchi (Fig 2, A). A suggestion of cylindrical bronchiectasis was also noted in the lower lobe. The lateral film (Fig 2, B), taken after some delay, disclosed these lower bronchi to be of normal caliber except for a peripheral branch of the posterior basal segment.

CASE 3—Bronchography was undertaken because of clinical suspicion of bronchiectasis. The immediate film (Fig 3, A) revealed incomplete filling of the left lower lobe bronchi without significant alteration in their proximal portions. By allowing further gravitation of iodized oil into the basal segments, the delayed film (Fig 3, B) disclosed both sacular and cylindrical bronchiectases, which might otherwise have been overlooked.

DISCUSSION

Although techniques for bronchography have been discussed in numerous articles,¹⁻⁶ no direct reference has been made to the indications for a delayed roentgenogram. Only Parmis⁴ recognized the value of additional films in the serial study of bronchial filling for inflammatory and neoplastic lung changes.

Assuming a satisfactory technique for instillation and distribution of contrast media through the various segments of the bronchial tree, there are four factors which influence filling of the peripheral bronchi:

1. *Physical Nature of Contrast Substance*—Both oil- and water-soluble contrast media are now available for bronchography. The oils have been in use since bronchography was originated by Forester in 1921.⁷ Only recently have satisfactory water-soluble substances been reported.⁸⁻¹⁰ It appears that

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some of the disadvantages of the oil media may be overcome by agents such as Ioduran B. However, the slow elimination of iodized oil allows for delayed studies of those areas which may initially show incomplete filling. Aqueous substances, being rapidly absorbed or easily diamed, do not lend themselves to such studies. Our references to the delayed roentgenogram in bronchography, therefore, assume the use of iodized oils, such as Lipiodol or Iodochlorol.

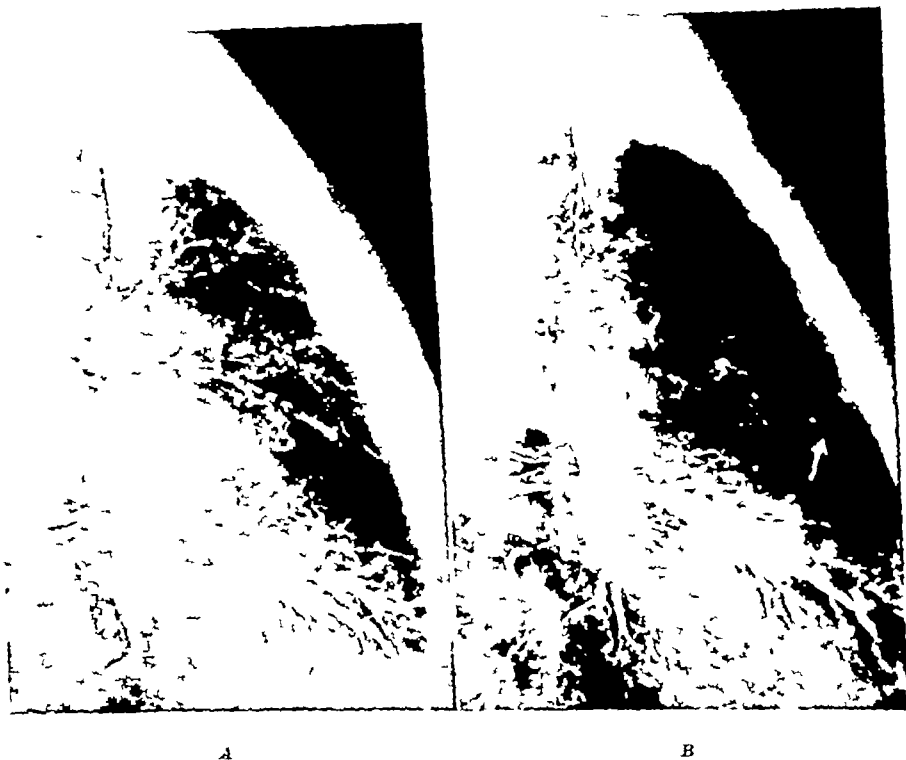


Fig 1—A Immediate film following bronchography. Right lateral view disclosing bronchiectasis in the right middle lobe. In addition the arrow indicates obstructed and dilated bronchus in the anterior segment of the right upper lobe. B Delayed film following bronchography. Similar film taken a few minutes later indicates obstruction of the bronchus in the right upper lobe to have been due to transient air block. The oil is seen to have penetrated to the periphery of this branch its caliber having returned to normal.

2 *Volume of Contrast Substance Used*—The influence of this factor on filling of the peripheral bronchi is an obvious one. The presence of an excess of iodized oil in the proximal bronchus excludes the question of inadequate contrast substance as a cause of incomplete visualization.

3 *Temperature of Contrast Media*—Reducing the viscosity of iodized oil by heating increases its speed of flow and its ability to enter stenosed bronchi. However, such changes in viscosity favor alveolar filling and make satisfactory distribution of the oil more difficult. Affecting reduction in viscosity of Lipiodol by dilution with a pure vegetable oil has the added disadvantage of decreasing its radiographic density. A short interval between the immediate and delayed

film allows the body tissues to affect warming of the oil with some reduction in its viscosity and favors gravitation into the peripheral bronchi, unless they are completely obstructed

4 *Patency of the Bronchi*—If we assume satisfactory control of the above factors, patency then remains the most important determinant in outlining the bronchi of any lung segment. Retained secretions, material trapped by stricture or by kinking of a bronchus and air blocked locally by the inflow of contrast substance, may each impose considerable, though transient delay in the passage of iodized oil. Incomplete information or uncertainty concerning



Fig 2—A Immediate film following bronchography. In addition to obvious saccular and cylindrical bronchiectasias in the left upper lobe, the bronchi in the lower lobe appeared to be obstructed and dilated. B Delayed lateral film following bronchography. Left lateral view disclosed the bronchi of the lower lobe to be of normal caliber except for a peripheral branch of the posterior basal segment. The appearance of the immediate film was apparently due to transient air block of the bronchi.

individual bronchi may result from such interference with their patency. The delayed film following bronchography helps to eliminate such transient sources of confusion.

A delayed roentgenogram is indicated when examination of the wet film taken immediately following bronchography discloses adequate contrast substance in a proximal bronchus, associated with failure to outline its peripheral branches. In the interval before taking such an additional film, the patient should be maintained, for about five minutes, in a position favoring gravitation

of the contrast substance into the unfilled segments. In the latter position he should be instructed to breathe very slowly and deeply, encouraging maximum dilatation and elongation of bronchi as a further aid to entrance of the iodized oil.

It may be seen from the accompanying illustrations (Figs 1, 2, and 3) that the delayed film following bronchography has proved of definite value by serving the following purposes: (a) avoiding the need for additional instillations of iodized oil or repeated bronchography due to incomplete filling of peripheral bronchi and (b) eliminating transient hydrostatic bronchial

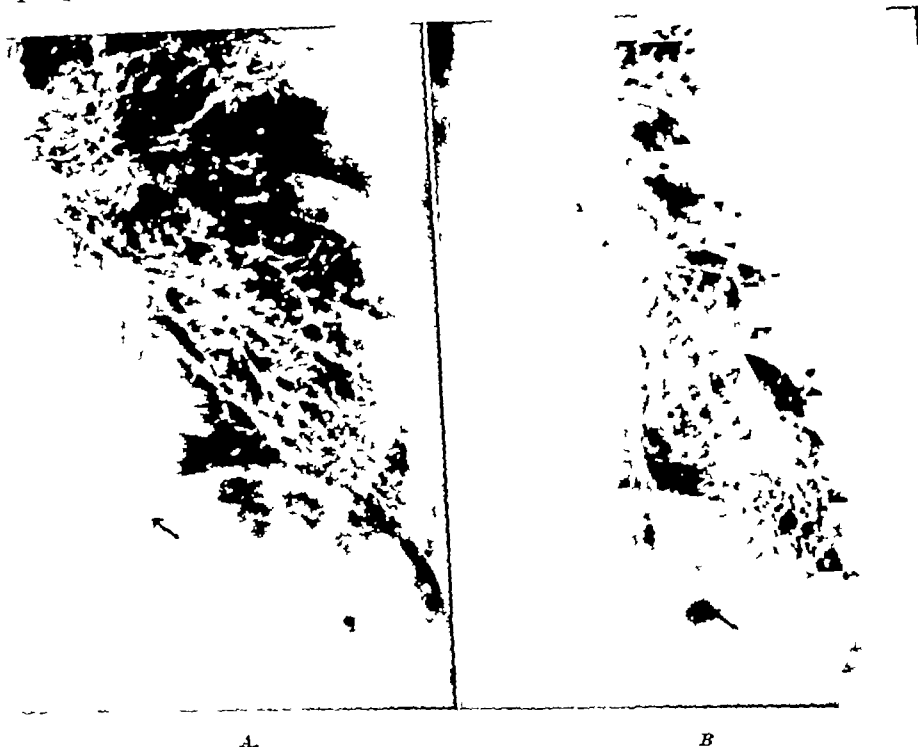


Fig 3—A, Immediate film following bronchography. Lower-lobe bronchi are seen through the shadow of the left ventricle. These appear obstructed the one indicated by the arrow being suggestive of minimal cylindrical ectasia. B, Delayed film following bronchography. A few minutes later the bronchus which appeared slightly dilated was seen to be of normal caliber. The more lateral bronchus visualized incompletely on the immediate film clearly shows cylindrical dilatation which might otherwise have escaped detection.

dilatations which might be mistaken for minimal bronchiectasis and needlessly included in resectional lung surgery or subsequently classified as "reversible."

The immediate roentgenogram following bronchial instillation of iodized oil remains essential to a clear demonstration of mucosal pattern, bronchial distribution and the interrelation of bronchi. Delayed roentgenograms following bronchography are, therefore, not intended to replace but rather to supplement the immediate films. The delayed roentgenogram will not obviate the need for expectorants, antispasmodics, postural drainage or bronchoscopy when these appear indicated as preliminaries to bronchography.

SUMMARY

The purpose and indications for a delayed roentgenogram following bronchography are outlined. Bronchograms are presented from three illustrative cases. Factors relevant to the subject of bronchial visualization are reviewed.

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ATRESIA OF CERVICAL ESOPHAGUS

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ATRESIA of the cervical esophagus from a fibromuscular stenosis of the cricopharyngeus muscle is extremely rare. The simple and effective method of the relief of the atresia makes this case worthy of reporting.

CASE REPORT

E R, 51 S366, a 12 year old schoolgirl, was admitted for the third time to St Mary's Hospital complaining of difficulty in swallowing for eight years. She was able to swallow liquids only in the past three years, and during the past year she would require twenty to thirty minutes to drink half a glass of water. Her swallowing was unchanged by emotions, fatigue, or diet. There was no associated coughing, choking or dyspnea.

Past history revealed a tonsillectomy at the age of 3 years, and fever of unknown cause six months later. Hesitancy in swallowing was noted about one year after tonsillectomy. Poor dental hygiene was noted, with extraction of upper incisors one year ago. No menses had occurred. The patient was about thirty pounds underweight.

Physical Examination—Temperature 98.8° F, pulse 84, respirations 22, body weight 39 pounds. The patient was malnourished and small of stature. Her head was well formed, upper incisors missing, tonsils absent, pharynx showed healed tonsillar fossa. Chest was symmetrical, no rales, heart tones were clear. Abdomen was soft, normal female genitalia, extremities symmetrical, reflex physiological.

Laboratory Data—Hemoglobin 10.9 Gm, red blood cells 4,060,000, white blood cells 12,890, urinalysis negative, Wassermann negative. Chest roentgenogram revealed no pneumonia, esophagram revealed atresia at cricopharyngeal region (Figs 1 and 2).

Hospital Course—Pharyngoscopy showed a stenosis at the pharyngo-esophageal junction that would barely admit a No 16 dilator. The mucosa was intact and normal. The left pyriform sinus was distorted in contrast to the opposite structure. The larynx was normal.

A Stamm gastrostomy was performed for feeding purposes and also for the retrograde introduction of radioopaque material in the lower esophagus. The study showed a normal esophagus up to the cricopharyngeus. No radioopaque material passed above this area. The patient was placed on a balanced gastrostomy feeding totaling nearly 2,200 calories a day. The patient remained at home for several weeks, and gained six pounds.

Upon readmission she appeared markedly improved in nourishment and growth. She received penicillin lozenges four times a day, and oral hygiene twice a day.

An exploration of the cervical esophagus was performed under gas oxygen ether anesthesia with an intratracheal catheter. A left sided semi collar incision was made, and the trachea and larynx mobilized. The left recurrent laryngeal nerve was retracted and the left inferior thyroid artery to the esophagus was divided so that the entire cervical esophagus and the lower third of the pharynx were exposed. The pharyngeus muscles joined together to form a sphincter at the cricopharyngeus. The sphincterlike muscle was approximately 5 by 9 mm thick, and the esophageal lumen was narrowed to 4 mm. It was well formed and rather firm in consistency. The sphincterlike band was easily freed from the subjacent longitudinal muscle fibers. An incision was made through the band with an immediate relaxation of the atresia area. A sensation of cutting fibrous tissue was experienced. Another incision was made through the band in the posterior

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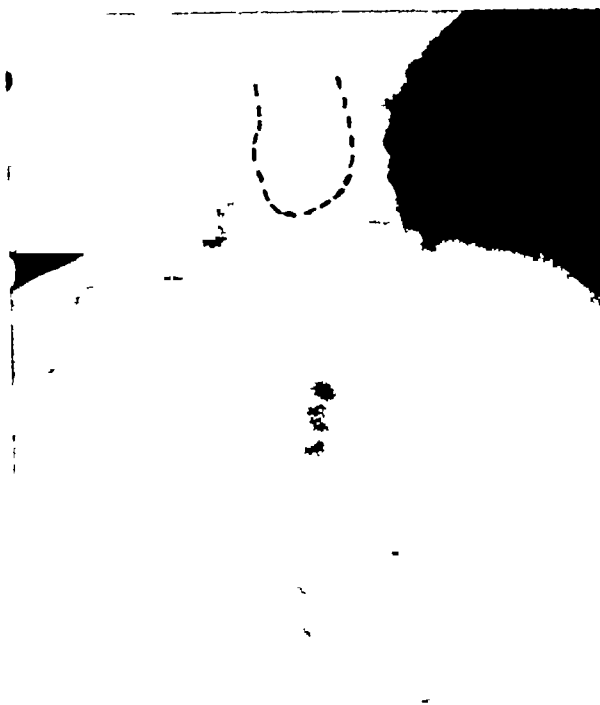


Fig 1 —Posteroanterior esophagram Barium failed to pass into the esophagus. Pharynx outlined



Fig 2 —Lateral esophagram showing barium outlining pharynx Esophagus occluded at the cricopharyngeal region.

portion. Thereafter the anesthetist was able to readily pass a No. 20F catheter. A portion of the sphincterlike band was excised for microscopic study. Slight separation of the longitudinal muscle elements exposed the mucosa which was intact and apparently normal. The cervical incision was closed in layers after placing a Penrose drain to the lowest part. An all silk technique was used throughout (Fig. 3).

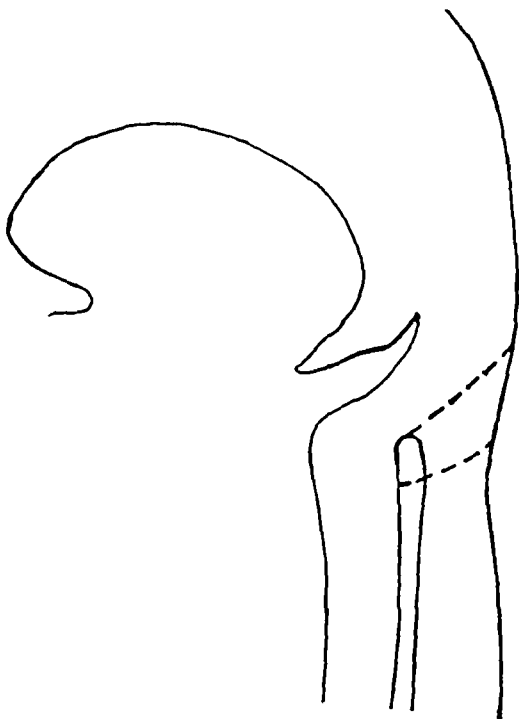


Fig. 3—Schematic outline of pharynx. Dotted line indicates cricopharyngeal band



Fig. 4—Low power photomicrograph of the fibromuscular band

The microscopic examination of the resected sphincterlike muscle band showed "normal striated muscle fibers. The blood vessels appear normal. There is no dense scar tissue. Scattered areas of fibrosis can be seen between muscle fasciculus" (Fig. 4).

Her convalescence was uneventful. There was a slight huskiness of her voice the first day, and on her third postoperative day she was ambulatory and beginning to take solid foods. She was discharged after gaining sixteen pounds.

A year later the patient had gained another twenty pounds. Fluoroscopic observation of a barium swallow failed to show any hesitancy. She states that she can eat all kinds of food rapidly and without distress (Fig. 5).



Fig. 5.—Postoperative esophagram. Barium passed readily into thoracic esophagus. Slight indentation in cricopharyngeal area but no obstruction.

COMMENT

The etiology of the stenosis may be a congenital atresia of the esophagus or a posttonsillectomy retropharyngeal abscess. The atresia is suggested by the intact and normal mucosa and the lack of paraesophageal fibrosis. Some difficulty in swallowing could have been present earlier but missed because of the young age of the patient. The clinical course, however, favors a low grade infection of the retropharyngeal area following a tonsillectomy with the development of fibrosis in the pharyngeus muscle and contraction of the cricopharyngeus muscle. The distortion of the left pyriform sinus would favor this view.

A preliminary gastrostomy was considered essential for improving the patient's nutrition. A more essential purpose was the attempt to determine the possibility of additional strictures in the lower portion of the esophagus.

Multiple strictures of the esophagus would cause one to favor resection with esophageal or esophagogastric anastomosis. A localized contraction would suggest a segmental resection of the esophagus with a direct anastomosis or the substitution of a skin-lined tube as in the Wooley procedure. The employment of the Heller procedure was strongly favored when it appeared that the mucosa was normal and intact. Heller's operation for cardiospasm of the lower esophagus was modified by freeing the sphincterlike band and resecting a portion. The procedure corrected the atresia effectively as evidenced by the bulging longitudinal muscle fibers through the created defect.

SUMMARY

A rare case of cricopharyngeal atresia is presented, with a simple highly effective corrective procedure.

EFFECTS OF INCREASED ENDOBRONCHIAL PRESSURE, VITAMIN A DEFICIENT DIETS, AND NASOPHARYNGEAL INSTALLATIONS OF BACTERIA ON THE TRACHEOBRONCHIAL TREES OF YOUNG RATS

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THE many theories regarding the causes of bronchiectasis have been capably reviewed by Andrus,¹ by Adams and Escudero,² Lisa and Rosenblatt,³ and by Mallory.⁴ Much experimental work has been done on the production of bronchiectasis, and the investigations and reviews of Weinberg,⁵ of Adams and Escudero,² and of Tannenbeig and Pinner⁶ are outstanding. The effects on the respiratory tract of complete and partial bronchial obstruction, atelectasis, infection, and variations in intrathoracic pressure have been studied by many workers.

In discussing case histories of patients with "idiopathic" bronchiectasis, clinicians have frequently commented on the following observations: (1) the onset of symptoms in the early years of life, as shown by Perry and King,⁷ 42 per cent of whose patients had the onset in the first decade of life, with 69 per cent of the patients developing the disease before the age of 20 years, (2) the frequency of the onset of symptoms of bronchiectasis following bronchopneumonia—pertussis and other respiratory infections have been discussed by Field,⁸ and (3) the same author has described the poor nutritional status of most of the patients with bronchiectasis.

It is of interest to note the two factors which are common to this condition, chronic cough and malnutrition, and to consider the possible relationship which such factors might have to the development of bronchiectasis.

Cough as a bronchodilating factor consists of two components: (a) the increased endobronchial pressure during the compressive phase of coughing immediately preceding the expulsive phase, and (b) the deep inspirations immediately preceding and following a cough. The physical factors active in coughing, and the various opinions regarding its relationship to bronchiectasis have been reviewed by Andrus.¹

Malnutrition may have two important sequelae, particularly in patients with bronchiectasis: (a) general decrease in resistance to infection, and (b) the specific effect on tracheobronchial epithelium resulting from inadequate vitamin A intake or utilization. This latter effect, as shown by Mori,⁹ by Wolfbach and Howe,^{10, 11} and by Wolfbach and Bessey,¹² consists of metaplasia and keratinization of normal epithelium, probably occurring first in the respiratory tract¹² but also involving the alimentary and urinary tracts as well as the eyes.

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and the paraculal glands. The resulting loss of ciliated respiratory epithelium has been considered a factor in decreasing the resistance of the respiratory tract to infection. The increased incidence of respiratory infection in vitamin A depleted animals has been discussed by Clausen,¹⁴ Robertson,¹⁵ and by Steiner, Greene, and Kramer.¹⁶ Respiratory infections, including bronchiectasis, occurring in many children with cystic fibrosis of the pancreas, have been described by Anderson,¹⁷ by Farber,¹⁸ and by Zuelzer and Newton.¹⁹ Many of these same children demonstrate metaplasia of the respiratory epithelium associated with vitamin A deficiency, which in turn is probably a sequel of the pancreatic disease.

Consideration was therefore given to any effect which chronic cough might have on bronchi whose epithelium had been altered by vitamin A deficiency, with and without the elements of induced infection. Attempts were made to study these effects experimentally in rats, and any relationship they might have to bronchiectasis. The element of bronchial obstruction was purposely avoided.

Rats were felt to be satisfactory animals for such a study because of ease of handling and production of vitamin A deficiencies, and because they do develop bronchiectasis. Wilens and Sproul²⁰ studied 457 rats of an inbred strain of pure albino rats of Osborn-Mendel stock, all of which had died spontaneously at average ages of 700 to 750 days, and found bronchiectasis and lung abscesses in 75 per cent of them. Saxton, Barnes, and Spelling²¹ studied the lung changes in rats of various ages, and did not find any bronchiectasis or chronic pneumonia prior to 250 days of age, with an increasing incidence of these diseases in rats above this age. All animals used in the studies to be reported were less than 250 days old at the time they were sacrificed.

EXPERIMENTS

Group I served as controls and received a regular diet consisting of the following:

Protein	24.8 per cent
Fat	4.6 per cent
Fiber	4.8 per cent
Carbohydrate	49.3 per cent
Nitrogen, free	44.4 per cent
Ash	9.5 per cent
Vitamin A	610-650

International units per 100 grams

Group II received a regular diet and was also subjected to increased endobronchial pressure of 10 centimeters of water for thirty minutes a day. The endobronchial pressure was created by using air-tight metal chambers with glass tops (Figs 1 and 2). The front wall of each chamber was made of rubber dam, having an opening in it of such size that when the rat's head was placed through the opening, the rubber dam fit snugly about the animal's neck and did not constrict or leak at the pressures used. Movements of the rat's body were controlled by placing the animal in a lucite tube fitted with metal clamps or opposed "V" shaped which could be adjusted about the animal's neck without constriction, but prevented the rat from moving his neck too far forward or backward. The chamber containing the rat's head was connected to

a source of air pressure, and the system fitted with a water manometer to allow for accurate adjustment of pressure, and also equipped with "safety" manometers to prevent pressure in any chamber exceeding the preset level. The anterior chest wall was removed from a rat and it was noted that this apparatus would increase endobronchial pressure during both inspiratory and expiratory phases and give some simulation of the cough mechanism. The pressure was set empirically at ten centimeters of water, and each rat was held in the chamber thirty minutes daily from 21 to 109 days (average, 72 days).

Group III received a diet deficient only in vitamin A (United States Pharmacopoeia XII, page 636). The following vitamin A deficient diet was used:

Vitamin free casein	18 per cent
Salt mixture	4 per cent
Dried yeast	8 per cent
Starch	65 per cent
Vegetable oil	5 per cent
Vitamin D	3 USP

Units per gram supplied by irradiated ergosterol

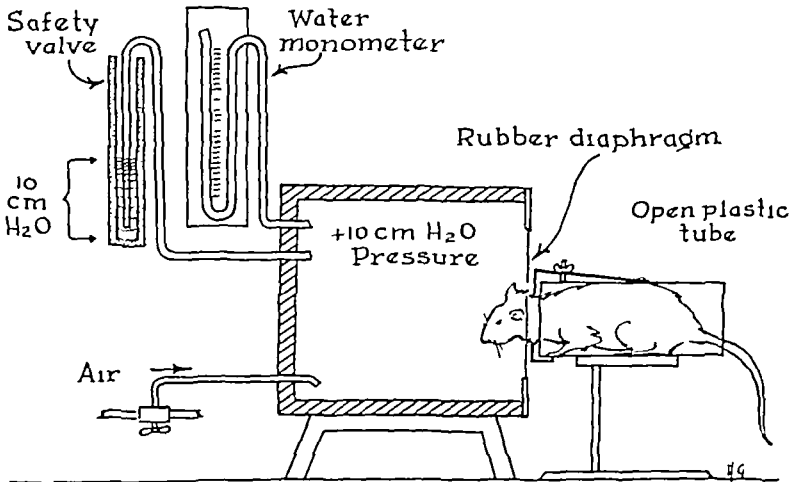


Fig. 1—Sagittal section diagrammatically showing arrangement of animal in lucite tube with the rubber diaphragm around his neck preventing loss of air from the closed metal chamber.

The air inflow tube, water manometer, and water safety valve maintain the +10 cm. of water pressure of air upon the animal's head.

Group IV received a diet deficient in vitamin A and was subjected to increased endobronchial pressure as in Group II.

Group V received a full diet and received one nasopharyngeal instillation by pipette, of 0.25 cc. of a suspension containing a 50 per cent pure culture of alpha streptococcus and 50 per cent staphylococcus.

Group VI was given a combination of a diet deficient in vitamin A plus the nasopharyngeal instillation of bacteria.

Group VII received a diet deficient in vitamin A, was subjected to increased endobronchial pressure, and nasopharyngeal instillation of bacteria.

In the vitamin A deficient groups the vitamin deficiency was established before the bacterial suspensions were instilled. Sixty to seventy-two days were allowed to establish the deficiency.

PATHOLOGIC CHANGES

When the animals died or were sacrificed, the trachea was immediately clamped and the lungs excised and placed in a 4 per cent solution of formalin. Sections were made at various levels through each lung and stained with hematoxylin and eosin.

Group I (15 rats, controls) The control group of young rats was sacrificed at average ages of 170 days, and on microscopic examination the bronchi of all were found to be normal. The lungs of three animals showed slight emphysema.

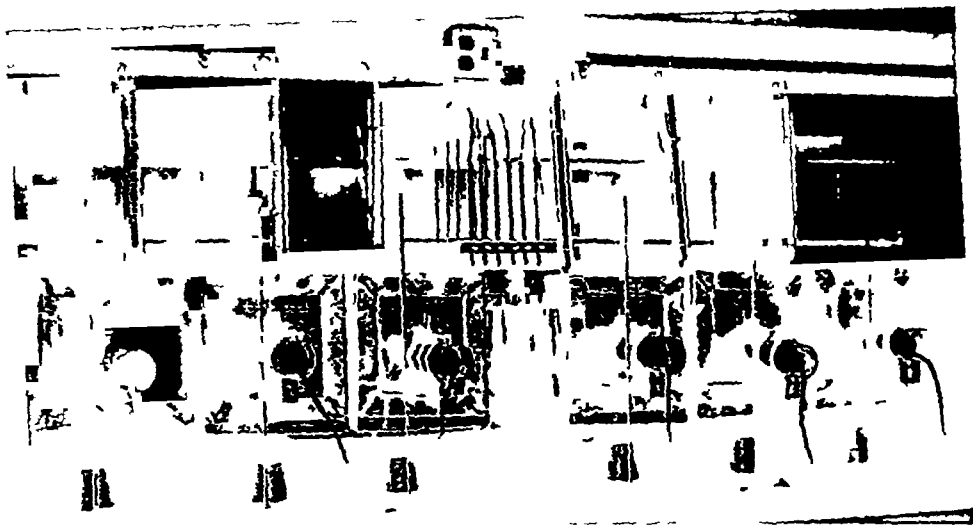


Fig 2—Use of a battery of lucite tubes and pressure chambers permits six animals to be exposed at one time to increased endobronchial pressures. A water manometer is seen above each chamber. The central glass jar with six tubes entering the water comprises the safety valve arrangement to prevent pressure in any chamber exceeding the pre set level of 10 cm. of water.

Group II (15 rats, regular diet plus increased endobronchial pressure for an average of 96 days) This group showed normal bronchi with no evidence of dilatation, but the lung tissue in all showed moderate emphysema and red blood cell congestion in the pulmonary blood vessels. The layers of the bronchial walls were normal, and showed no changes which might be attributed to the increased pressure.

Group III (15 rats, vitamin A deficient diet for an average of 79 days) All animals showed metaplasia of the cylindrical or cuboidal type epithelium to the squamous keratinizing type, which was attributed to the vitamin A deficiency. The fibrous and muscular tissue of the bronchial walls was clearly demarcated, with no abnormality noted. Eight of the rats showed normal

bronchi and seven showed dilated bronchi. Thirteen of the group demonstrated slight to moderate emphysema and red blood cell congestion in the alveoli. Two rats showed infiltration of neutrophils in the bronchial walls along with exudate and inflammatory changes in the lung parenchyma justifying a diagnosis of bronchitis and pneumonia in each.

Group IV (15 rats, vitamin A deficiency established and increased endobronchial pressure for an average of 66 days). Fourteen rats showed metaplasia of the respiratory epithelium. The fifteenth animal did not demonstrate it, due probably to insufficient time (25 days) on the vitamin A deficient diet. Eleven rats showed normal bronchi and four revealed dilated bronchi, although evidence of bronchiectasis was not present in the bronchial walls. All showed emphysema and red blood cell congestion in the pulmonary alveoli in Group II.

Group V (18 rats, regular diet and intranasal bacteria for an average of 92 days). Bronchitis was found in only one rat in this group and there were no other significant findings.

Group VI (19 rats, vitamin A deficiency established and intranasal bacteria for an average of 87 days). Again there was evidence of pulmonary emphysema and red blood cell congestion in alveoli in all animals. In this group there were four animals showing evidence of bronchitis, five demonstrated pneumonia and six rats developed pulmonary abscesses. There was no evidence of bronchiectasis.

Group VII (15 rats, vitamin A deficiency established, increased endobronchial pressure, and intranasal bacteria for an average of 83 days). All animals showed pulmonary emphysema plus red blood cell congestion in the alveoli. Vitamin A deficiency was produced in all of the rats of this group before the bacterial suspension was introduced, and 30 to 45 days were allowed to elapse after bacterial instillation before the animals were sacrificed. There was no evidence in this group of bronchitis, pneumonia, pulmonary abscess, or bronchiectasis.

DISCUSSION

In young healthy rats, metaplasia of the respiratory epithelium, increased endobronchial pressures and bacteria instilled into the respiratory tract, did not, either singly or in various combination, under the conditions of the experiments, produce any changes in the bronchi or lungs which could be interpreted histologically as bronchiectasis. It does not rule out the possibility of achieving different results by varying the degree or duration of endobronchial pressures used, the duration of dietary insufficiency, or the quality or quantity of bacterial instillations. However, any results obtained in rats more than 250 days old would be difficult or impossible to interpret. Goldblatt and Bemischek²² have found that vitamin A deficiency alone may produce infection and abscess in the bronchioles of rats due to blockage by the keratinized cells which have been shed from the walls. Pulmonary infections then may occur from the bacteria already normally present at the site, bacterial growth being favored by poor drainage, dead tissue and the lack of ciliary action and metaplasia.

TABLE 1

GROUPS	NO OF RATS	BRONCHII NOT DILATED	DILATED BRONCHII	EMPHY- SEMA	RED IN GOOD CELL CON- GESTION	BRON- CHITIS	1 NEU- MONIA	1 UL- MONARY ABSCESS	VITAMIN A DE- FICIENCY	BRONCHII ECTASIS
I Regular diet (controls)	15	15	0	3	0	0	0	0	0	0
II Regular diet and in- creased endobronchial pressure	15	15	0	15	15	0	1	0	0	0
III Vitamin A deficient diet	15	8	7	14	14	2	2	0	15	0
IV Vitamin A deficient diet and increased endo- bronchial pressure	15	11	4	15	15	0	0	0	14	0
V Regular diet and inso- pharyngeal bacteria	18	16	2	4	0	1	0	0	0	0
VI Vitamin A deficient diet and nasopharyngeal bacteria	19	16	3	13	17	4	5	6	19	0
VII Vitamin A deficient diet, increased endo- bronchial pressure and nasopharyngeal bacteria	15	8	7	15	15	0	0	0	15	0

and mucus secretion which are the sequelae of the metaplasia. Two animals in Group III that were on the vitamin A deficient diet alone showed bronchitis and pneumonia but no evidence of bronchiectasis.

All rats subject to increased endobronchial pressure did develop some degree of pulmonary emphysema, even though rats in all other groups, not subject to increased endobronchial pressures, also showed varying incidences of emphysema (Table I). Such findings would tend to support the opinion expressed by Andrus¹ that the forces active in coughing may be so distributed as to cause pulmonary emphysema.

Of the three groups of animals receiving bacterial installations, the group on normal diet showed the least change, only one animal developing evidence of bronchitis. In the group with vitamin deficiency alone, more changes occurred following installation of bacteria than in the group with vitamin deficiency plus elevated endobronchial pressures. Why such a difference should occur is not clear. Tannenbeig and Pinnet,⁹ using rabbits, introduced infectious material into the bronchi distal to the point of bronchial ligation, and demonstrated severe bronchiectatic changes in the lung distal to the bronchial obstruction, while the opposite lung showed extensive bronchopneumonia.

In all animals used here, the element of bronchial obstruction was purposely avoided, and the absence of bronchiectasis in these rats would tend to lend evidence in support of the theory that complete or partial bronchial obstruction, probably plus infection are essential to the production of bronchiectasis. The findings would further tend to favor the opinions of Fairber¹⁸ and of Zuelzer and Newton¹⁰ that in the production of bronchiectatic changes in the lungs of children with cystic fibrosis of the pancreas, the blockage of bronchioles by thick, tenacious mucus is probably a more important factor than the metaplasia of the bronchial epithelium.

SUMMARY

1 Metaplasia and keratinization of the bronchial epithelium could be produced in young healthy rats by the use of vitamin A deficient diet.

2 The effects of the three following factors: (a) metaplasia of bronchial epithelium, (b) elevated endobronchial pressure (10 cm. of water elevation for thirty minutes daily over average periods of 72 days), and (c) nasopharyngeal instillation of bacteria (0.25 cc. of a suspension of equal parts of alpha streptococci and staphylococci introduced once) were studied in young healthy rats, singly and in various combinations. Various degrees of pulmonary emphysema were produced, along with a few instances of bronchitis and lung abscesses but there were no histologic changes in the lungs of any of the animals to suggest that bronchiectasis had been produced.

3 The absence of bronchiectasis in the rats studied would seem to lend evidence in support of the theory that complete or partial bronchial obstruction, probably plus infection, are essential to the production of bronchiectasis.

The authors wish to express their grateful appreciation to Dr. Grant Abley and Edward Patterson for their assistance in this work.

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TRANS-STERNAL TRACHEAL EXCISION FOR CARCINOMA

REPORT OF A CASE

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THE successful treatment of obstructive lesions of the mid-trachea is rendered difficult because of the presence of the overlying great vessels and sternum. Heretofore, for the management of tracheal neoplasms we have had to rely on the incomplete endoscopic removal of the endotracheal portion of the growth or on the doubtful effect of roentgen therapy on lesions of a type which commonly show little response. The following case report demonstrates the feasibility of applying the principles of surgery for malignant lesions to this part of the body. Only one similar instance could be found in the literature.¹

J. S. B., a 62 year old retired white rural mail carrier, first noted hoarseness about Dec. 1, 1948, ten weeks before admission, following an upper respiratory infection. This hoarseness continued with a "raw" feeling in the throat and slight discomfort on swallowing. The patient's general health was excellent. He had become slightly deaf in the last few years, there had been the usual childhood diseases, he had had repair of inguinal hernia in 1912 and an appendectomy in 1937. He was a heavy smoker and an occasional heavy drinker. Otherwise his history was negative.

Laryngoscopic examination under topical anesthesia with pontocaine was performed on Feb. 9, 1949, at which time a whitish tumor mass was found which was situated on the right vocal cord, occupying its entire length and jutting into the posterior commissure. A biopsy of this lesion revealed it to be epidermoid carcinoma, Grade I II. The patient was admitted to the University of Virginia Hospital on Feb. 17, 1949. On physical examination he was found to be a slender but well developed elderly white man. The only significant findings were moderate hypertrophy of the tonsils, a few shotty cervical nodes and a moderate systolic hypertension. The blood Wassermann and Kahn tests were negative. The urine was negative. The blood urea was found to be 17 mg per cent. The plasma protein was 6.7 Gm per cent. The hemoglobin was 13 Gm per 100 cubic centimeters (84 per cent), and the red blood cell count 4.45 million per cubic millimeter. The white blood cell count was 13,000 per cubic millimeter, and the differential count was normal.

On Feb. 18, 1949, a total laryngectomy was performed under anesthesia with intravenous sodium pentothal supplemented by local infiltration of 1 per cent procaine. The pathologist reported "Epidermoid carcinoma of the larynx, Grade I II, involving the left vocal cord, the left elastic cone, and the posterior commissure." The patient had an uncomplicated convalescence, and left the hospital on March 1, 1949. He returned on March 9, 1949, with labored respirations, and a crust of inspissated mucus was found and removed, with immediate relief. On repeated check up examination he was found to be doing well except for slight crusting of the tracheostomy, until Nov. 17, 1949, when a slightly piled up gray elevation was noted 1 cm within the tracheal ostium, on the patient's left and anteriorly. Biopsy was reported as epidermoid carcinoma. On Dec. 1, 1949, five radon seeds were implanted anteriorly after removal of recurrent neoplasm with cup biopsy forceps.

On Jan. 19, 1950, visible tumor tissue was seen projecting into the trachea from its anterior wall 1 cm. within this area, which discharged a small amount of serosanguinous material. He was admitted the following day for roentgen therapy which was administered between

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Jan 21, 1950, and Feb 7, 1950, to a total of 3,400 r over the upper sternum, suprasternal notch and tracheostomy site with the following factors 200 kv, FSD 50 cm, 25 Ma, H.V.L 1 mm. copper. Roentgenograms of the chest showed a moderate amount of emphysema with some flattening of the diaphragm. The aorta contained much calcium, and the radon seeds implanted Dec 1, 1949, were visualized as small opacities overlying the fourth dorsal vertebra. There was no evidence of osseous or parenchymatous metastases. At the time of discharge on Feb 8, 1950, there was a marked hyperemia of the skin in the treated areas and some recession of the visible tumor. On March 30, 1950, there was some limitation of the airway, which had progressed by April 27, 1950, to the point where the laryngectomy tube could not be inserted. By May 11, 1950, the tracheal stenosis had become marked and the patient was again admitted. Examination showed him to be using accessory muscles of respiration. The tracheostomy site was surrounded by a slightly elevated rim of firm tissue. The neoplasm was visible within the ostium as a grayish red, granular, firm projection from the lateral walls, particularly the right, which extended down the trachea for a distance of 3 to 5 cm and reduced the airway to a vertical slit 2 mm in width. No cervical or supraclavicular nodes were palpable, and the tracheostomy and recurrence were freely movable. Roentgenograms of the chest showed no change from the previous study of March 30, 1950.

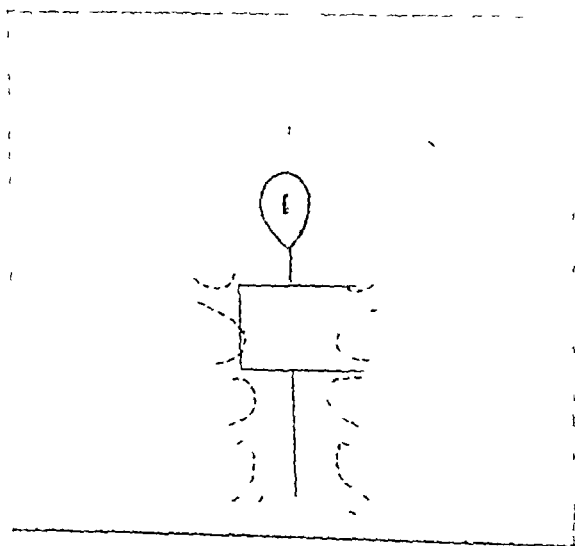


Fig 1—Outline drawing of skin incision.

The thoracic consultant felt that "Although operation may be highly dangerous because of the extensive irradiation, a *laissez faire* policy will certainly be fatal within a few weeks. Since there is no evidence of metastases and since asphyxia is inevitable, we feel that a tracheoplasty through the divided sternum may remove the neoplasm and restore the airway." On May 15, 1950, an anterior mediastinotomy with removal of the upper trachea and trans sternal tracheoplasty was carried out. The operative note except for an initial paragraph containing a brief history and the indication for operation, follows:

"Anesthesia was induced with intravenous pentothal, following which a rather small intratracheal tube was inserted through the tracheostomy opening and anesthesia continued with ether oxygen. The anterior surface of the neck and chest was prepared and draped. A rectangular skin flap was outlined over the manubrium, with its base about the level of the left first interspace. This was dissected up and preserved with a saline

sponge, with the aim of constructing a skin tube if the remaining normal trachea should not be long enough to reach the skin over the sternum. An incision was then extended over the midsternum and downward nearly to the xiphoid and upward to encircle the tracheostomy (Fig. 1). The suprasternal notch was cleared of areolar tissue between the heads of the sternocleidomastoid muscles and the index finger inserted into the anterior mediastinum. The sternum was split longitudinally with the Lebsche knife. The thyroidea ima vein was divided between ligatures close to its entrance into the left innominate vein. This latter structure was later divided between ligatures and suture ligatures to expose the innominate artery and left common carotid artery as far down as the point where they originated from the arch of the aorta. The trachea was located in the V-shaped cleft between these arterial structures. The intratracheal tube was now removed, the trachea thoroughly suctioned, and a sterile intratracheal tube reinserted on

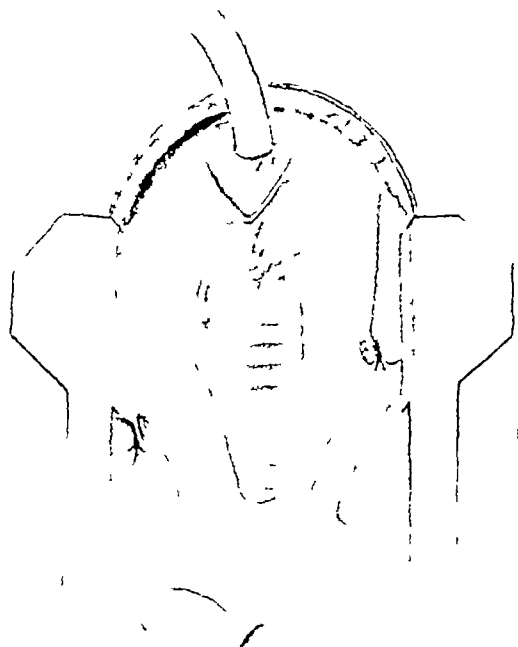


Fig. 2.—Sketch showing the relationship of the tumor-bearing upper trachea to the neighboring vascular structures

the operator's side of the screen. This was connected by rubber tubing to the anesthetic machine over the top of the screen. This allowed the tracheostomy to be encircled and the tumor-bearing portion of the upper trachea was gradually freed of its surrounding fibrous and muscular attachments (Fig. 2). The right lobe of the thyroid was cut away from its attachment to the upper trachea which was then carefully elevated and dissected free posteriorly from the inferior hypopharynx. It was felt that during this dissection all the tumor was removed. The tumor apparently extended for about 5 cm. down the trachea from its external opening. On the right lateral side of the trachea just below the thyroid two large succulent nodes were dissected out with the trachea which was now elevated between the innominate artery and the left common carotid artery almost as far down as the bifurcation. Semicircular openings in the lower manubrium were rounded away so that when the sternum should be pulled together, an adequate opening

through its central portion would be present to allow the emergence of the trachea to the skin (Fig 3). At a point about 1 cm below the lower portion of the recurrent carcinoma, the trachea was attached on both sides to the inner periosteum of the sternum with interrupted sutures of No 36 steel wire. The tumor containing portion was now an



Fig 3—Drawing of the mobilized trachea after ablation of the tumor and preparation of the semicircular openings in the manubrium



Fig 4—Appearance of patient six weeks after tracheal excision.

puted. From this point on, anesthesia was given intermittently by inserting a clean intratracheal tube into the trachea. The prepared flap of skin on the left was approximated to the trachea with interrupted double zero chromic catgut sutures over two thirds of the circumference of the trachea on the left. The sternum was now drawn together with a few interrupted No. 30 steel wire sutures in the anterior periosteum and a flap of skin prepared on the right side which was then used to complete the skin tube through the sternum. An attempt was made to close the dead space previously occupied by the upper portion of the trachea and the skin was closed in layers above and below the new tracheostomy. Penrose drains were left in the substernal space and in the upper medias tinum at the base of the neck. Two areas left bare of skin were then covered with split thickness grafts taken from the anterior thigh, a No. 8 laryngectomy tube was inserted into the new tracheostomy and a dry dressing applied to the wound. The patient tolerated this extensive procedure quite well and left the operating room with a clear airway and in excellent condition."

The report of the Department of Pathology read as follows: "A tubular section of the trachea which measured 5 cm. in length and 3 cm. in diameter. Seven cartilaginous tracheal rings are included. The surface is covered with grayish fibrous adhesions and attached to one side near the distal end is an ovoid soft grayish nodule 1 by 1 by 1.5 cm. The wall of the trachea varies from 3 to 7 mm. in thickness and includes several areas of firm grayish white tissues suggestive of carcinoma. The mucosal surface is slightly wrinkled and gray beneath which are friable, necrotic appearing pockets 4 to 5 mm. across, which contain three yellow radon seeds. Following a microscopic description the final diagnosis was recurrent epidermoid carcinoma of the trachea with negative lymph nodes, adequately excised."

The patient was given antibiotics and high caloric tube feedings, and was ambulatory on the first postoperative day. His course was satisfactory until May 26, the wounds healing nicely and the grafts showing a 90 per cent take. On that date, the thoracic resident's note says: "The trachea seems adequately anchored. There is some slough of the skin edges and considerable sticky exudate." On May 31, the wound was inflamed, and purulent exudate emerged from between the divided sternum. The airway was satisfactory, although there was considerable crusting of the upper trachea. The patient was kept in a small room with a croup kettle, but he still complained of dryness of the air and himself arranged a protecting framework of wire covered with flannel which he placed over the tracheostomy and kept moist with water.

By June 28 he had learned to care for the tracheostomy, which was now freely patent without the tube. The wound had improved greatly in appearance, and drainage was not more than 15 to 25 cc. daily (Fig. 4). The temperature was usually between 99° and 100° F. He was discharged at this time but returned July 3, 1950, inebriated. There had been progressive improvement of the wound and it was felt that the sternum eventually might heal completely. He was discharged July 8, 1950.

Efforts were made to have this man return for check up examinations, but no more was heard of him until the report of his death Sept. 30, 1950, at home from what was apparently a sudden large hemorrhage from within the substernal space.

SUMMARY

This report describes the recurrence following total laryngectomy for epidermoid carcinoma of a similar lesion within the tracheostomy. Whether this was an extension, an implantation, or the occurrence of a totally separate primary cannot be determined. The surgical treatment of this lesion and its sequelae have been described.

REFERENCE

1. Watson, William L.: Cancer of the Trachea Fifteen Years after Treatment for Cancer of the Larynx. *J. THORACIC SURG.* 12: 11-150, 1941.

ENDOBRONCHIAL CARCINOSARCOMA

H E TAYLOR, M D, AND M V RAE, M D
VANCOUVER, B C

THE term carcinosarcoma denotes a mixed tumor containing the elements of carcinoma and sarcoma. Whether such an entity actually exists is a matter of controversy.

When faced with such critical reviews as those of Saphir and Vass⁶ and Willis,⁷ one hesitates to use this term in classifying a neoplasm. However, we wish to report two identical and unusual endobronchial tumors which show a similar histologic appearance, being composed predominantly of spindle cells simulating a fibrosarcoma intermixed with which are scattered islands of obvious squamous cell carcinoma. These tumors appear to be quite distinctive, and in our opinion should be separated from the usual bronchiogenic carcinomas.

CASE REPORTS

CASE 1—A white man, aged 69 years, complained of a persistent dry cough, general malaise, and fever of two months' duration. He claimed to have aspirated a cough drop during sleep, following which he had tightness in the chest and dyspnea. There was no hoarseness, chest pain, or hemoptysis, but his cough and dyspnea increased. He had always lived a healthy outdoor life and had no medical consultations except for a gunshot wound of the arm in World War I and a prostatectomy eleven years previously. He was a heavy cigarette smoker and used alcohol infrequently. Physical examination showed restricted movement of the right side of the chest with dullness to percussion and diminished breath sounds which were almost absent over the base. The trachea was shifted slightly to the right.

Roentgenogram—There was atelectasis of the right lower and middle lobes.

Laboratory Examinations—Vital capacity was 1,500 cc, white blood count 10,700 per cu. mm, sedimentation rate varied from 71 mm to 109 mm per hour (Westergren), sputum was negative for acid fast bacilli and for tumor cells, cultures gave a growth of *Streptococcus hemolyticus*.

A bronchoscopy was performed and revealed the right main bronchus to be plugged with brownish necrotic tissue considered to be a bronchogenic carcinoma. A biopsy from this showed only chronic inflammatory and necrotic tissue interpreted as being related to the aspirated cough drop. Following penicillin, a further bronchoscopy was performed seven days later and showed a red fungating tissue filling the right main bronchus. Several portions were removed, weighing in all 45 grams. Sections of this tissue showed a tumor of sarcomatous appearance consisting of interlacing fasciculi of coarse spindle cells with large nuclei, prominent nucleoli, many mitoses, and some tumor giant cells. This was interpreted as a sarcoma, possibly of neurogenic origin. Pneumonectomy was performed three months after the onset of his symptoms. No pleural adhesions were found, and the middle lobe was atelectatic.

Surgical Specimen—This consisted of the entire right lung, the pleural surfaces were smooth. The upper lobe was aerated, the middle and lower lobes were atelectatic. In the major bronchus was found an elongated cigar-shaped pedunculated soft rubbery, grayish red tumor 4 by 1.5 by 1 cm. It was entirely endobronchial, and its only attachment was

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a small short pedicle from its midportion to the lower lobe bronchus, the tumor extending, both up and down from the pedicle, its upper limit covering the bronchial aperture to the middle lobe but did not enter it. The lower extremity formed a firm plug in the lower lobe bronchus beyond which pus filled the lumen.

Microscopic Examination—The main part of the tumor lay within the bronchial lumen, but the tumor pedicle protruded through an intercartilaginous space forming a small buttonlike knob with a discrete condensed capsulelike border lying against normal lung alveoli. Much of the surface was necrotic and ulcerated (Fig 1). The tumor was very cellular and consisted of two distinct elements, fibrous stroma and squamous epithelial islands, both of which showed active growth and were intermixed. The main bulk of the tumor consisted of broad sheets of spindle cells with fasciculation (Fig 2). There was much variation in size and shape with fusiform spindle cells, short oval cells, and stellate cells with long processes (Fig 3). The nuclei were large, oval, pale, and vesicular, with prominent single and multiple nucleoli. Numerous pyknotic nuclei and bizarre mitoses



Fig 1—(Case 1) Low power view to show entire tumor. The elongated upper portion was largely fibro-sarcomatous (Figs 2 to 4) with occasional areas of carcinoma the latter being predominant in the base (Figs 5 and 6). (Hematoxylin and eosin $\times 4$)

were evident, with multilobed nuclei and occasional tumor giant cells. Sections impregnated with silver showed distinct reticulin fibrils intimately associated with these cells (Fig 4). The blood channels were fine slits lying adjacent to the tumor cells. In the base of the tumor were areas of obvious squamous carcinoma embedded in the mesoblastic tissue (Fig 5). These areas were small circumscribed, well demarcated, and often separated by a slitlike space from the stroma. Stratification of cells was well marked, with some cornification and pearl formation. A few areas were less differentiated and here the cells were larger, darker, more pleomorphic, and immature, with atypical mitoses. These latter areas were less discretely separated from the stroma and in a few areas tended to blend very intimately with the stromal cells (Fig 6). The bronchial lymph nodes were enlarged and hyperplastic but there was no invasion by tumor.

Follow up—The patient was discharged six weeks after operation, and three years later is still well complaining only of dyspnea. There is no evidence of recurrence or metastases.

CASE 2—A white man, aged 57 years, consulted his doctor because of increasing dyspnea, pain in the chest, and occasional hemoptysis of some two months' duration. He had lost about 20 pounds in the previous month. Examination revealed diminished expansion of the left chest with dullness to percussion and absent breath sounds. Radiologic examination showed atelectasis of the left lung, probably due to bronchial obstruction. Bronchoscopic examination revealed a mass in the left major bronchus, and a biopsy was taken. Microscopically, however, this showed only mucus with a small piece of bronchial

Fig 2.

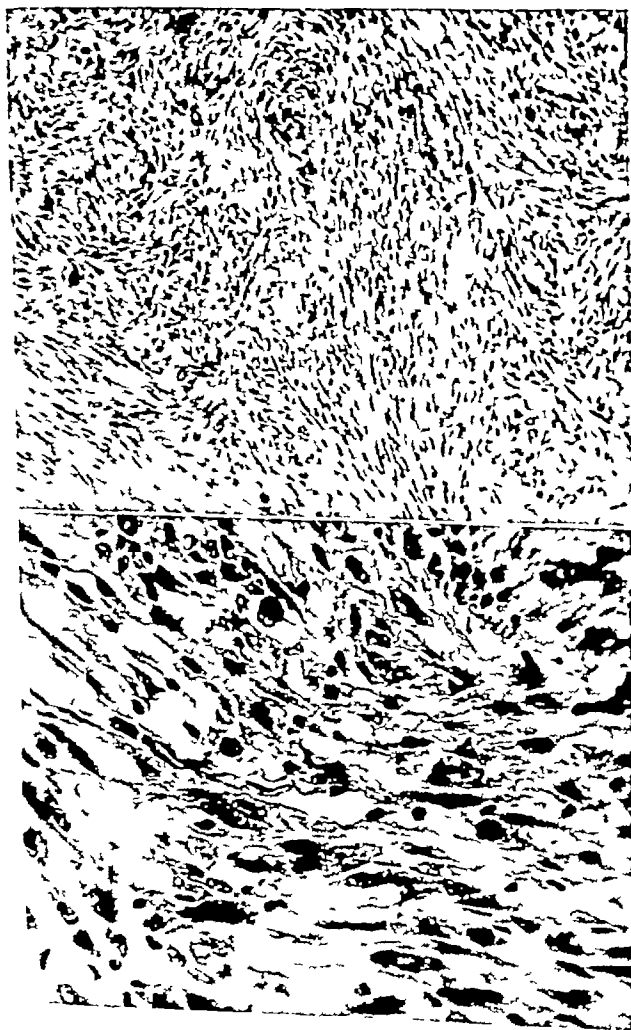


Fig 3

Fig 2.—(Case 1) Fibrosarcomatous area of neoplasm showing interlacing fasciculi of spindle cells (Hematoxylin and eosin $\times 100$)
 Fig 3.—(Case 1) High power view of an area from Fig 2 to show cell details and fibrils (Hematoxylin and eosin $\times 400$)

mucosa, no neoplastic tissue being present. Seventy two hours later he suddenly became dyspneic and shocked. He expectorated large quantities of blood and, in spite of resuscitative measures, he failed to rally and expired eight hours later.

Necropsy Findings—Thorax The pleural cavities were dry, and no adhesions were present. The trachea contained thin frothy blood stained fluid. The left major bronchus was completely filled with clotted blood, forming a cast. When this was removed there was found a pedunculated tumor arising from the posterior wall of the left main bronchus just proximal to the bifurcation of the upper and lower branches. It measured 2.0 by 1.0 cm., and was attached to the wall of the bronchus by a wide base. On sectioning the tumor appeared to be entirely endobronchial, there being no extension into the adjacent lung parenchyma. **Lungs** The lungs showed acute edema, and there was an acute suppurative bronchiolitis distal to the tumor. The bronchial and mediastinal lymph nodes showed no gross tumor infiltration. The remainder of the necropsy showed no further lesions.

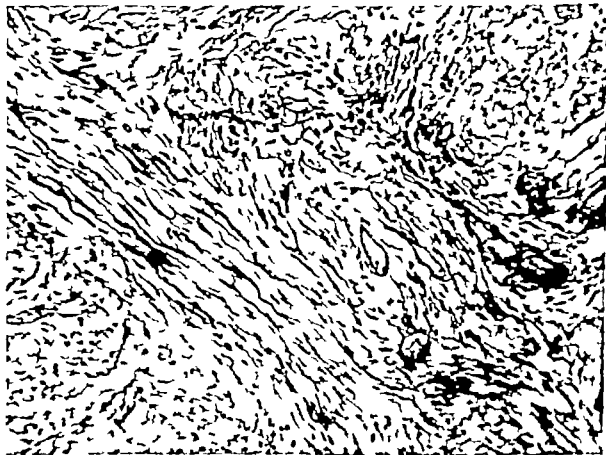


Fig. 4—(Case 1) This shows the marked reticulin formation by the spindle cells in the fibrosarcomatous area of the neoplasm. (Silver Impregnation Wilders method $\times 100$)

Microscopic Examination—Bronchial tumor This had a pedunculated appearance similar to Fig. 1, the main tumor projecting into the lumen of the bronchus. The vast majority of the neoplasm was very cellular simulating a sarcoma, being composed predominantly of spindle cells, many having irregular bizarre nuclei (Fig. 7). In many areas the cells were arranged in bands and interlacing fasciculi. In other areas they had a haphazard arrangement with, here and there, a larger tumor giant cell. Masson stains showed delicate collagen fibrils intimately associated with the spindle cells (Fig. 8). At the base of the neoplasm between the cartilaginous plates, the picture was quite different. Here were nests of typical squamous cell carcinoma (Fig. 9), most of which were well differentiated, many of the clusters showing polygonal keratinized cells with an occasional small epithelial pearl. Many of the nests lay in a space, an occasional one being lined with flattened endothelial cells suggesting lymphatics. Separating these neoplastic masses was dense, acellular collagen. Serial sections failed to show any transition between the obvious carcinoma and the cellular sarcomatous areas. Lymph nodes and all other tissues failed to show any evidence of metastatic tumor.

DISCUSSION

These two tumors are characterized by an admixture of apparent fibrosarcoma and squamous cell carcinoma. In Case 1 a few areas were found in which the carcinomatous tissue faded into the sarcoma, suggesting that the

latter might be a bizarre spindle cell variant of squamous carcinoma. It is well known that such a transition may be seen, but in most instances this type of tumor is quite obvious. Such is not the case here, for if one views the spindle cell areas of these tumors alone, one is forced to interpret these regions as fibrosarcoma, the neoplastic cells showing a distinct reticulin and collagen fibril formation. Further, in Case 2 we were unable to show in serial sections any transition between the carcinoma and the spindle-cell areas. Some of the bi-

Fig 5



Fig 6

Fig 5—(Case 1) An area from the base of the tumor showing islands of squamous carcinoma. Here the sheets of carcinoma cells are separated from the stroma by clear spaces. (Hematoxylin and eosin $\times 100$)

Fig 6—(Case 1) Another area showing less differentiated squamous carcinoma with variations to spindle cells. The latter however are dissimilar to the sarcomatous areas depicted in Figs. 2 and 3. (Hematoxylin and eosin $\times 100$)

zant tumor giant cells interspersed in the sarcomatous areas do, however, have an epithelial appearance and probably represent an admixture of carcinomatous cells in the predominantly sarcomatous regions.

Saphir and Vass,⁶ in their classical review of 153 cases designated as "carcinosarcoma," accept only three or four examples as true instances. The majority they interpret as examples of primary carcinoma showing areas in which the carcinomatous cells have assumed a spindle character simulating sarcoma. Seven of the cases reviewed by them involved the lung, but they accepted only one instance of a mucinous adenocarcinoma with associated spindle and pleomorphic cells as a possible example of carcinosarcoma. Willis, while accepting examples of carcinosarcoma in animals, knows of no indubitable example of this neoplasm in human material. Foulds,² in his review, offers several explanations to explain complex tumors with multiple neoplastic components including an intermingling of independent primary tumors, the possibility of simultaneous

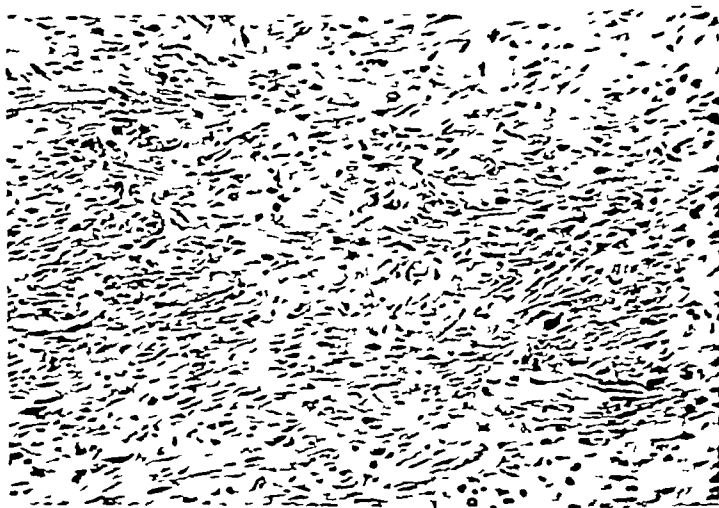


Fig. 7.—(Case 2.) Fibrosarcomatous area of tumor showing bizarre spindle cells with hyperchromatic irregular nuclei. The main mass of the tumor had this appearance. (Hematoxylin and eosin, $\times 120$.)

neoplasia in two closely associated tissues, e.g., parenchyma and stroma, or neoplasia of stroma or invaded tissues in a primarily simple tumor. Foulds unfortunately does not give my clear cut opinion of his own on this subject, his paper being primarily a review in which he largely cites other authors' opinions.

On the other hand Harvey and Hamilton⁴ have suggested that carcinosarcomas might result from sarcomatous change in the stroma of a carcinoma. More recently Bergmann, Ackerman, and Kemler⁵ have reported two tumors that appear to be identical to those described here, and they have interpreted them as true examples of carcinosarcoma. In a review of the literature they consider eight other reported tumors from the lung to belong in this group and a further four to represent collision tumors. They also cast some doubt upon the high rejection rate of carcinosarcomas by Saphir and Vass.

We have been unable to find examples of similar tumors other than those included in the review by Bergmann and his associates. Rock and Hall have reported a case of squamous-cell carcinoma of a bronchus and spindle cell sarcoma of the lung, but this they interpreted as multiple primary tumors rather than a true carcinosarcoma.

Fig. 8

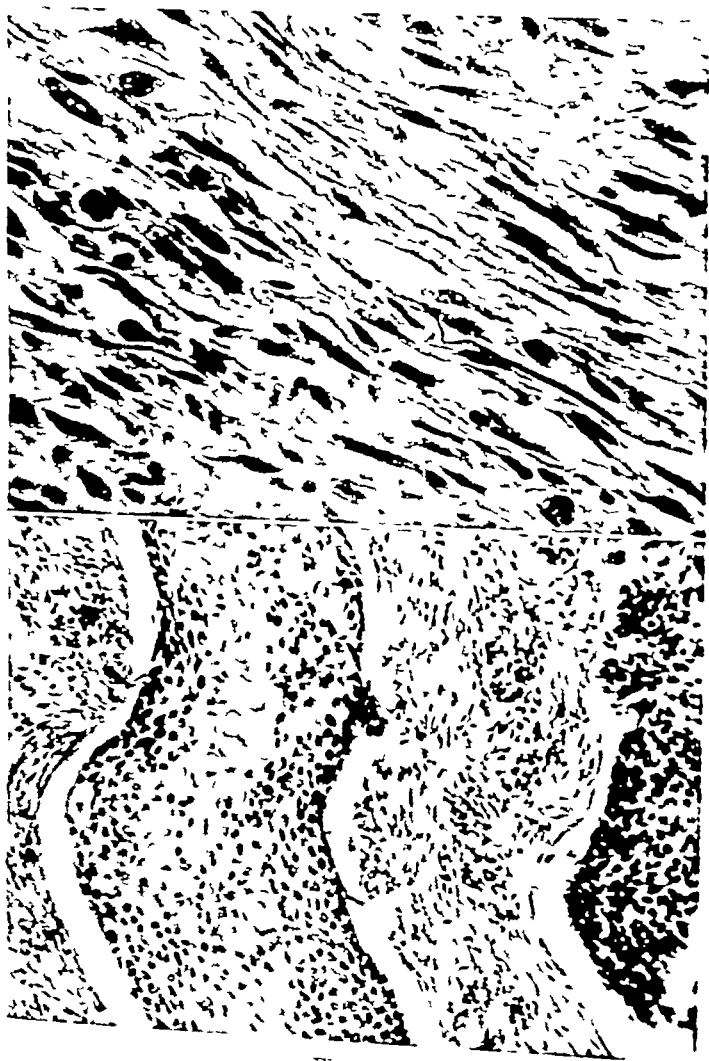


Fig. 9

Fig. 8—(Case 2) High-power view of an area from Fig. 7 to show collagen fibrils being formed by spindle cells (Masson's trichrome stain $\times 400$).
 Fig. 9—(Case 2) An area from the base of the tumor showing squamous carcinoma. The stroma here is not neoplastic. (Hematoxylin and eosin $\times 120$).

In view of the distinctive histologic appearance of these tumors we are of the opinion that they should be classed separately as endobronchial carcinosarcomas. Furthermore their clinical behavior and prognosis appear to warrant

this separation. One of our cases is living and well three years after pneumonectomy, while one of Bergmann's cases has survived six years and the other one year and seven months at the time of the report. In this respect they resemble sarcoma of the lung. As Iverson⁴ has stated, "The patient is equally fortunate in having a sarcoma of the bronchus or its imitator carcinosarcoma in lieu of a classical bronchogenic carcinoma."

SUMMARY

1 Two distinctive endobronchial tumors have been reported, composed of spindle cell fibrosarcomatous areas with intermixed squamous cell carcinoma.

2 These are interpreted as true carcinosarcomas rather than squamous cell carcinomas showing transition to a spindle-cell variant.

3 These tumors appear to have a better prognosis than bronchiogenic carcinoma. One patient is living and well three years after pneumonectomy. The other, who died of massive hemorrhage following bronchoscopic biopsy, showed no evidence of metastases or extension to the lung at necropsy.

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WOUND OF THE SUPERIOR VENA CAVA WITH SURVIVAL

THEODORE R HUDSON, M D
CHICAGO, ILL

WOUNDS of the great vessels in the mediastinum are so deadly that there are almost no recorded cases of such wounds with survival. Prior to the present time there has been but one record of a patient who survived a wound of the superior vena cava. In 1933 Biggers¹ reported the case of a patient who had been stabbed in the right chest, the blade entering near the sternum at the level of the third intercostal space, with rapid development of symptoms of cardiac tamponade. A diagnosis of probable wound of the right auricle was entertained, and the patient was explored through an anterior incision. The pericardial sac was tense with blood and bore a small rent over the region of the right auricle. This rent was enlarged, and a large gush of blood occurred. Digital pressure within the pericardium controlled the active bleeding, and a wound of the intrapericardial portion of the superior vena cava was eventually located. It was successfully closed with interrupted sutures, and the patient survived.

The case reported here is different in that the tear in the vena cava occurred above the pericardial reflection, and was located in such a position as to section the phrenic nerve. The effect of this difference was that it permitted a much greater immediate loss of blood with the dangers inherent to exsanguination and excessive mediastinal shift.

CASE REPORT

A 43 year-old woman was hit at virtually point blank range by a bullet from a .38 caliber pistol. The slug entered the right chest just below the first rib cartilage, immediately to the right of the sternum. It emerged posteriorly just to the right of the spine at the level of the sixth posterior rib. When brought to the hospital* at 2 A M she was desperately short of breath and in a state of shock. Physical examination revealed two cleanly drilled bullet holes in the location described above. There were dullness and absent breath sounds over the entire right hemithorax, and the apex beat of the heart was shifted far to the left. Portable roentgenogram of the chest showed a complete blank out of the right lung and a great shift of the mediastinum to the left. Also shown was complete section of the sixth rib near the spine with tiny fragments of lead left clinging to the ends of the broken bone (Fig 1).

Whole blood, plasma, and vasopressor drugs were quickly given to combat the threatening state of shock and gradually, over the course of a few hours, the condition of the patient became relatively stable so that she was reasonably comfortable with oxygen administration. Some eighteen hours after the injury she was seen in consultation. She was very dyspneic and apprehensive. There was a large quantity of blood in the bed, the result of leakage from the posterior wound of exit. An attempt was made to aspirate the pleural space with a large needle, but this was impossible due to clot formation. When asked to cough, as she was sitting upright, a gush of air and blood came from the posterior wound. At this point the blood pressure was falling and it was obvious that thoracotomy was indicated.

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*Dennor Hospital St. Charles, Ill. Service of Dr. Norman J. Schreiber

[illegible]

Pls. I ~ I would like to know if you have any more information on this matter. I am sure you will be able to find out what happened to the man who was taken to the hospital. I am sure you will be able to find out what happened to the man who was taken to the hospital. I am sure you will be able to find out what happened to the man who was taken to the hospital.

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the lung and trapped the air out of the pleural space. The patient was returned to her room in much better condition than when she left it. Her convalescence was uneventful except for a temperature of 105° F on the second postoperative day. This was not explained, since there was no clinical nor laboratory evidence of transfusion reaction, and

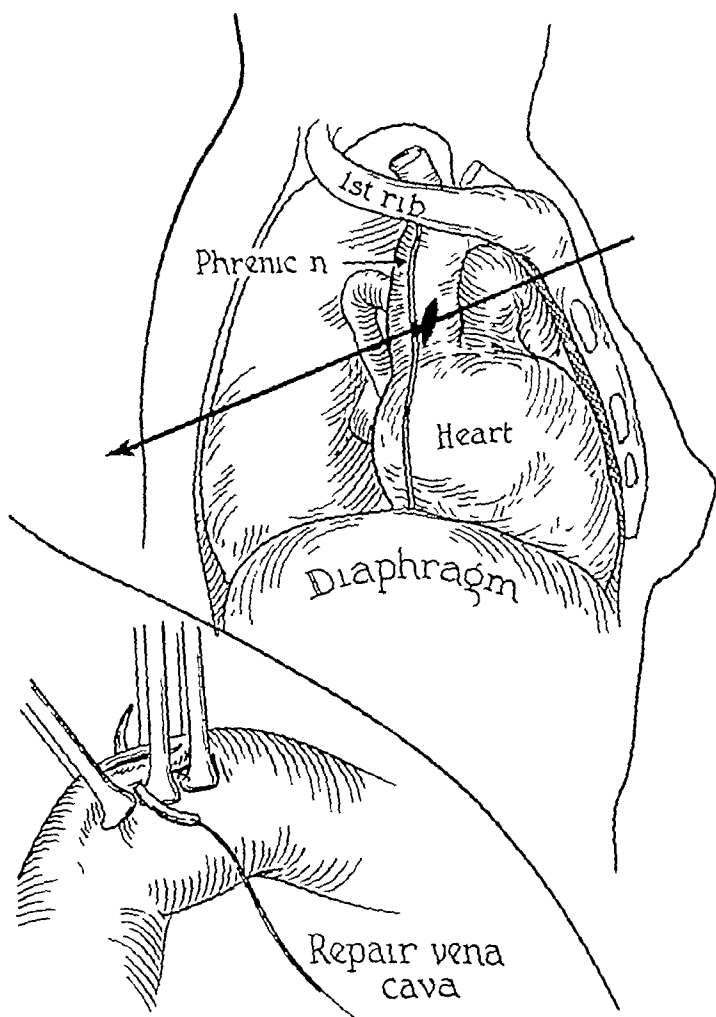


Fig 2—Wound in the superior vena cava with section of phrenic nerve. Insert Method of repair. Artist has shown interrupted suture whereas actual closure was done with running suture.

since she always raised her pulmonary secretions quite well. She was discharged on the tenth day after operation. Fluoroscopy on the day of discharge from the hospital showed total paralysis of the right leaf of the diaphragm. A roentgenogram taken two weeks later (Fig 3) showed the right lung well aerated, and a marked rise of the right diaphragm.

COMMENT

Certainly wounds of the superior vena cava will practically always be fatal. It seems likely that the fortuitous clot formation on the wound in the

vessel saved this patient's life. The rapid filling of the right pleural space no doubt brought about a state of equilibrium between the increased intrapleural pressure and the lowered pressure in the vena cava so that the clot could form presenting a barrier to further bleeding. It is interesting to speculate, in retrospect, what would have happened had the pleural blood *not* been clotted so that aspiration could have been done. It seems very likely that, with reduction of pressure in the pleural space by the withdrawal of fluid blood, the clot would have blown off allowing further, and doubtless fatal, hemorrhage.



Fig. 3.—Chest film two weeks after injury showing marked rise of right diaphragm following section of phrenic nerve by bullet.

SUMMARY

A case of gunshot wound of the superior vena cava and section of the phrenic nerve with survival is reported.

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EXPERIMENTAL METHODS FOR DETECTION OF CHANGES OF BLOOD SUPPLY TO THE HEART

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LOS ANGELES, CALIF

THE introduction of surgical procedures to produce an auxiliary blood flow to the myocardium for coronary artery disease has posed new problems. O'Shaughnessy's¹ omentopexy was designed to bring blood to the ischemic myocardium from the mesenteric circulation by transplanting the omentum to the surface of the myocardium. Beck's² pectoral transplant and intrapericardial scarification combined with instillation of talc or asbestos was calculated not only to bring auxiliary blood supply via the pectoralis muscle but also to produce vasodilatation of the coronary circulation and thus to increase its natural collateral circulation.³ Thompson's^{4, 5} cardiopericardiopexy utilized asbestos particles inserted into the pericardium to increase natural collateral circulation. Beck's^{6, 7} operation of transplanting a systemic artery into the coronary sinus was designed to bring systemic blood flow as an auxiliary source of blood to the myocardium via retrograde flow through the coronary venous system. Vineberg⁸ implanted the internal mammary artery into the myocardium thus producing auxiliary blood flow directly to the muscular channels of the myocardium. Ligation of the coronary sinus was designed to increase the arterio-venous collateral coronary circulation.⁹ Beck's newest two stage operation appears to be most promising. In the first stage a 3 to 4 mm sized connection between the ascending aorta and the coronary sinus is made with a vein graft. A few weeks later at the second operation the coronary sinus is stenosed with a

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Nylon ligature This operation brings arterial systemic blood with a high pressure gradient into the venous system of the myocardium¹⁵ It would appear that arteriovenous anastomoses would be a necessary pathway for such retro grade flow to effectively reach the myocardial capillary system

Thus the surgical procedures so far have been concerned with

- 1 Irritating the myocardium so as to produce vasodilatation and vascular intrapericardial adhesions
- 2 Bringing new systemic arterial blood supply to the surface of the myocardium with pedicled omentum, pectoralis muscle or lung
- 3 Bringing arterial blood supply directly to the myocardium or coronary sinus

The chief problem concerning these surgical procedures is how to evaluate their effectiveness as auxiliary collateral coronary circulations To do this, methods for detection of changes in the myocardial blood supply are necessary since ischemia is the chief process being corrected by the surgically induced collateral circulation

All these surgical procedures have been designed to increase the normal physiologic collateral circulation of the myocardium The tests of benefit from the newly acquired circulation have almost exclusively been worked out on dogs¹⁶ In dogs, surgically induced auxiliary vascular supplies to the myocardium have been shown to exist by anatomic studies¹⁶⁻¹⁸ Physiologically, bleeding experiments from the distal cut end of the coronary arteries after ligation of the proximal end, and survival experiments following coronary ligation, have indicated the functional state and effectiveness of the surgically induced collateral coronary circulation¹⁶ It is hoped that such surgically induced additions to the normal myocardial collateral circulation may protect against coronary insufficiency and further ischemia associated with effort, blood pressure changes, and additional infarcts

To better understand the problem of evaluating the effectiveness of surgically induced, auxiliary collateral blood supply to the coronary circulation we shall now review in brief some of the experimental observations made in this laboratory which show the nature of the collateral coronary circulation in normal and ischemic hearts of both dog and man¹⁹⁻²¹

- 1 By the use of saline suspension of erythrocytes labeled with radio active phosphorus, the existence of numerous anastomoses in the left ventricle and of a collateral circulation between the right and left ventricles was demonstrated in the normal post mortem human heart
- 2 By the use of measurable glass spheres ranging in diameter from 10 to 400 micra the largest intercoronary arterial anastomosis in the normal human heart was found to allow the passage of spheres ranging from 70 to 180 micra These findings indicate that such intercoronary arterial anastomoses are of arteriolar dimensions in the normal heart

- 3 Arteriovenous coronary anastomoses were demonstrated by recovery of glass spheres 70 to 170 micra in diameter from the coronary sinus after their injection into the right or left coronary arteries
- 4 The diameters of the anastomotic channels between the coronary arteries and either of the ventricular cavities range from 70 to 220 micra. These channels represent one or all of the following vessels: arteriololuminal, arteriosinusoidal, and Thebesian veins
- 5 By intravenous injection of labeled erythrocytes into moribund patients with normal and ischemic myocardiums, the presence of a functioning collateral circulation to all parts of the myocardium was demonstrated including the infarcted areas
- 6 From the observations with intravascularly injected erythrocytes it was concluded that the collateral circulation to the myocardium of the dog, which had been rendered ischemic by ligation of the anterior descending coronary artery, supplies all parts of the ischemic region, furthermore this collateral supply of blood circulates actively throughout the ischemic area, but the pericardial side of ischemic myocardium of the left ventricle is better nourished by the collateral circulation than the endocardial side
- 7 In shock not only is the general systemic blood pressure lowered but also the coronary blood pressure. Under conditions of shock the collateral circulation to an area of the myocardium, made ischemic by ligation of the anterior descending coronary artery, has little or no function since the blood pressure is too low for pumping blood efficiently through the collateral circulation
- 8 The reverse was also shown to be true. If the blood pressure was raised by transfusion of heparinized blood, the collateral circulation to an ischemic area was restored

The preceding discussion has described changes in the collateral coronary circulation that can be found in all normal dogs' beating hearts. Just as ingestion of carbohydrate elicits an increased output of insulin, the production of myocardial ischemia calls forth an increase of collateral coronary circulation. The problem is how best to detect and evaluate that additional collateral blood supply induced by experimental surgical procedures. For obvious reasons, it would appear desirable to have visual tests which may be performed upon the dog's beating heart rather than to depend upon findings in dead hearts. Likewise, it is desirable to have tests which have already had a base line of experimental detection of normal coronary collateral circulation. It is the purpose of this paper to present three visual methods for detecting changes of blood supply to the dog's beating heart. These tests are (1) fluorescein, (2) contractility, and (3) electrical activity.*

*A colored motion picture was shown to illustrate the three tests

FLUORESCEN

Prinzmetal and associates¹³⁻¹⁵ have shown previously that the coronary circulation can be visually demonstrated in the dog's beating heart by slow motion pictures (50 to 100 frames per second), photographed under near ultra violet light to allow the intravenously injected fluorescein to become visible within the coronary circulation. The following observations are pertinent to the use of fluorescein as a test of benefit from the additional collateral circulation which might be supplied to the myocardium by the particular surgical procedure.

- 1 In animals with normal blood pressure, fluorescein appeared in the coronary arteries rapidly, 4 to 7 seconds, and in the next few seconds the entire heart became intensely fluorescent.
- 2 If the dye were injected directly into only one artery, the anterior descending artery of a dog's beating heart, the dye could be visualized in the coronary circulation of an area many times greater than that normally supplied by the anterior descending coronary artery.
- 3 It was visually demonstrated that the coronaries were not end arteries and that there was physiologic coronary collateral circulation in the normal dog's myocardium detectable by the fluorescein method.
- 4 In shock, induced by hemorrhage, diminution of the coronary blood flow could be demonstrated since the appearance time and intensity were delayed but all the coronary circulation eventually visualized.
- 5 When fluorescein was injected into the femoral vein after the mid anterior descending artery was ligated in dogs with normal blood pressure the fluorescein rapidly filled the ischemic region from contiguous areas.
- 6 The ischemic regions generally became intensely fluorescent 1 to 2 seconds after the control regions.
- 7 If the blood pressure were lowered to shock levels (20 to 50 mm. of Hg mean pressure) the appearance time in the infarcted ischemic areas was markedly prolonged or even absent. The blood pressure was too low to pump the fluorescein through the collaterals and so the infarcts did not visualize with fluorescein.
- 8 The reverse was also shown to be true if a dog (in shock with coronary artery ligated) had his blood pressure restored with transfusion of heparinized blood, the fluorescein test showed the ischemic area to fill from contiguous areas through the normal collateral circulation.

By the fluorescein test the ischemia or shock and of experimental coronary infarction was readily detected. From the observations above listed it can be concluded that there is an always ready normal natural collateral coronary circulation which however has limited effect upon myocardial ischemia. Probably small internally occluded muscle masses are rendered relatively normal and completely protected. But larger ischemic muscle masses may re-

sult in central necrosis of the myocardium when the collateral circulation is not adequate. Furthermore persistence of nonfilling, central cyanosis of the infarcted area during fluorescein test indicates that no collateral circulation is effective for this area. The normal predicted response for the physiologic collateral circulation after ligation of the anterior descending artery of a dog's beating heart would be only a second or two delay of the fluorescein into the ischemic area. It is suggested that additional collateral blood supplied by experimental surgical procedures ought to be detected by this method. If the new blood supply were efficient it should allow fluorescein to fill areas made ischemic by coronary ligation with but slight delay in appearance time since the new blood flow would not be interrupted by the coronary ligation. If the additional blood flow from the operative procedure were 100 per cent efficient, the predicted visualization with fluorescein would approach that of the normal contiguous areas and the muscle mass supplied by the ligated coronary artery should not appear ischemic. For these reasons we feel that the fluorescein can be offered as a test of the benefit which might be derived from experimental surgically induced auxiliary collateral coronary circulation.*

CONTRACTILITY

Ischemic myocardium ceases to contract within 3 to 4 seconds after the anterior descending coronary artery is ligated.^{22, 23} In a dog's beating heart this cinematographic phenomenon of noncontractility of the muscle mass supplied by the ligated anterior descending artery is called "ballooning." Sometimes this phenomenon occurs only in late systole, but in other instances the ischemic region balloons during the entire systolic phase. Ballooning after ligation of small arteries may come and go, or even not occur at all, but larger ischemic areas balloon more consistently. After inducing hemorrhagic shock in dogs with coronary infarcts, the following significant changes occurred as a result of the low blood pressure in the slow motion colored movies

- 1 The ballooning became much more extensive in area and degree
- 2 Cyanosis of the ischemic area became marked
- 3 If ballooning had disappeared spontaneously, it reappeared and became more marked.
- 4 If there was only late systolic "ballooning" and not full systolic "ballooning," it now occupied the full phase of systole

The reversibility of "ballooning" could be induced by transfusing with heparinized blood and restoring the blood pressure from shock levels to normal. Such transfusions resulted in restoring the noncontractile "ballooning" to visibly normal contractions in the cinematographic observations. Not only did the "ballooning" cease or become less intense but the cyanosis also diminished or disappeared as the blood pressure was restored to normal. The observation that the "ballooning" is associated with myocardial ischemia can also be substantiated by the fluorescein test which shows slow or absent filling in the "ballooning" areas.

*Colored photographs from the slow motion movie with fluorescein are published in the American Heart Journal, Vol 35 page 18 May 1948. See reference No. 19

These experiments show that "ballooning" is associated with myocardial ischemia since it occurs after experimental production of ischemic infarcts. Furthermore the "ballooning" appears to vary with the effectiveness of the collateral coronary blood flow. Therefore "ballooning" should be suitable as a test of benefit for detecting the effectiveness of additional artificially supplied blood from surgical procedures. Efficient surgically produced collateral blood supplies should protect against the phenomenon of "ballooning" when myocardial ischemia is experimentally produced by ligation of the anterior descending coronary artery in the dog's beating heart. Less suitable and less efficient surgical procedures would probably not prevent the occurrence of "ballooning" after experimental coronary ligation. For these reasons we feel that "ballooning" can be offered as a test of benefit.

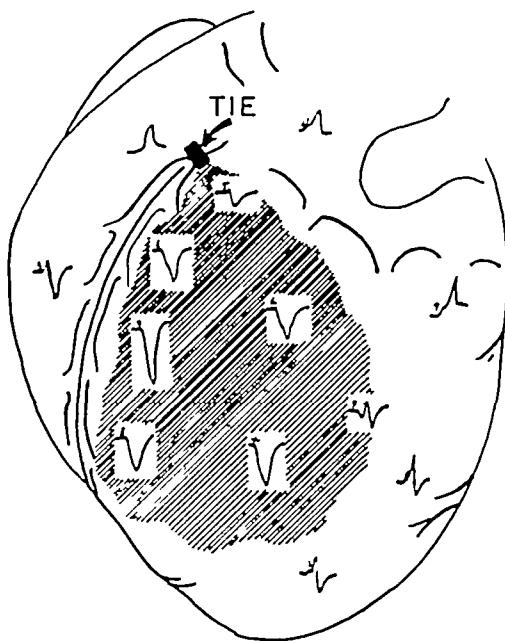


FIG. 1.—Diagram taken from data showing electrical activity in a dog's beating heart with a through and through infarct of two months duration. Tie is on the anterior descending coronary artery. Shaded area is the grossly ischemic region. Within the shaded area the explored spots record a Q wave. At the edges where there is less ischemia QR waves are recorded in the left ventricle and over the normal left ventricle R and RS waves are recorded.

ELECTRICAL ACTIVITY

Since the electrical activity of the myocardial contraction is related to the efficiency of the coronary circulation an electrocardiographic test of benefit induced by surgically made auxiliary collateral coronary circulation should be feasible. To study this problem experimentally, the anterior descending coronary artery in sixty dogs was ligated. These operations were performed through an intercostal approach with a 4 inch incision in the left fifth interspace. The dogs were prepared with morphine (0.03 Gm. per kilogram) and atropine (0.0004 Gm. per kilogram) in suitable doses and anesthesia was main-

tained with intravenous sodium pentothal. Continuous ventilation of the lungs was made through a semirigid intratracheal catheter and the portable Waters-Guedel anesthesia apparatus. One hundred per cent oxygen was the ventilating gas administered by intermittent manual compression of the 3 liter rubber bag*. The wound was closed air tight with the lungs fully inflated in order to avoid leaving any appreciable pneumothorax. Postoperatively one or two intramuscular injections of penicillin-streptomycin mixture (400,000 units of the former and 1 Gm. of the latter) were administered daily for one or two days. Although practically all the ligations were performed at a relatively high level, the mortality was approximately 15 per cent from immediate ventricular fibrillation. Only one delayed mortality occurred from empyema.



Fig. 2.—Enlarged still photograph from a high-speed colored movie picture frame. In this experiment the exploring electrode has a knob and is to the left and it records the lower tracing. Being over a normal area it records an upright positive R wave. The tiny silver electrode in the left ventricular cavity is just to the right and it records on the upper tracing a cavitary Q wave.

After various intervals, up to six months the surviving animals were then prepared for electrocardiographic studies. The chest was opened bilaterally and the sternum was partially removed together with suitable portions of the ribs. Anesthesia was morphine and intravenous urethane, and the lungs were ventilated continuously with 100 per cent oxygen with a suitable artificial pump and an intratracheal catheter. Although in some instances the pericardium was not opened, most of the electrocardiographic tracings were made on the exposed myocardium after suturing the opened pericardium to the chest wall. Electrical activity was recorded with a double channel high-speed pen writing Brush recorder at 125 mm. per second. The exploring electrode recorded the electrical activity directly from the surface of the myocardium or any desired spot.

*Anesthesia was administered by Dr. David Sobin and the operative assistant was Dr. L. Alfred Breckler.

Electrocardiographic recording was performed with the aid of a unipolar system using the Wilson central terminal. Routinely, as many as 40 or 50 chosen spots representative of both ventricles and the ischemic infarcted areas were explored (Fig 1). A special silver-needle, insulated, reference electrode, made so as to record only from its tip, was plunged into the left ventricular cavity in



Fig. 3—Same experiment as Fig. 2 of an electrocardiographic exploration of the surface of a dog's beating heart with a two month old through and through infarct. The exploring electrode is now over a fibrous area and it records on the lower tracing a Q wave almost the same as the cavity Q wave recorded on the upper tracing.



Fig. 4—The exploring electrode is now moved over an area in the infarct with a fibrinous texture. A heart and fibrinous ventricular mural mass and it records a Qtr wave in the lower tracing with the reference electrode continuing to record a cavity Q wave.

order to record simultaneously the left ventricular cavity potential. Simultaneously, the dogs beating heart and the electrocardiographic tracings were photographed in color at high speed so that visible contractility and electrical activity appeared in the same frame when the movie was projected. Although pathologic studies were made of all hearts, and special studies were also carried out for purposes of determining the exact origin of the electrocardiographic tracings obtained, only the Q, R, and QR wave changes will be reported here.

In infarcts of one or two months' duration, the surface was whitish on the pericardial side. Often myocardial contractility was visibly diminished over these areas. Over the normal areas of the left ventricle, not expected to be supplied by the anterior descending artery, usually normal red in color, and with good contractility, a positive deflection of an R or RS wave was consistently recorded (Figs 1, 2, and 5). Over the center of such infarcted whitish areas, or where no contractility in the whitish areas was present, a Q wave was recorded

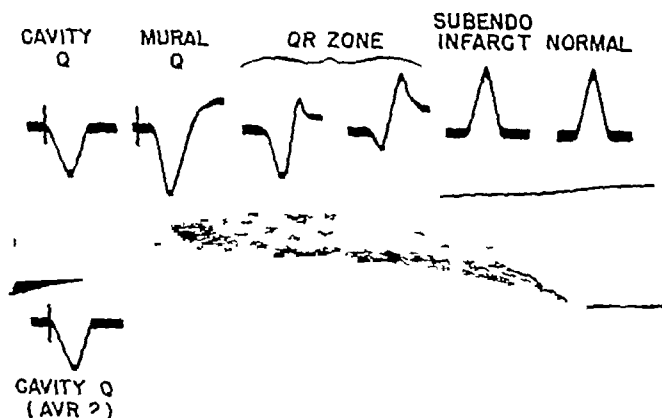


Fig 5—Diagrammatic representation of the type of electrocardiographic tracing obtained from the surface of the dog's beating heart correlated with the amount of infarction and its location. The completely shaded area represents through and through infarct and successively less involvement proceeding from left to right until only a subendocardial infarct and finally normal muscle is encountered. It can be seen as one proceeds from left to right, there is less Q and more R until the normal area is reached.

(Fig 3). As the exploring electrode was moved toward the periphery of the infarct from its center, less ischemic areas were encountered where QR waves were recorded (Fig 4). Over old infarcts with complete replacement of myocardium by fibrous tissue the exploring electrode recorded Q waves with the same configuration as the Q waves recorded by the cavity electrode (Fig 3). The histologic examination of the infarcted areas confirmed the finding that the more fibrous tissue replacement in the myocardium, the more Q and less R wave was present (Fig 5). In infarcts where the myocardium had not become ischemic "through and through," various quantities of muscle tissue may still be viable and contractile. In these areas of less ischemia, QR waves are often present (Figs 1, 4, and 5).

It would appear that measurement of electrical activity of the dog's beating heart by the method described is suitable for detecting ischemic areas of infarct following ligation of the anterior descending coronary artery. This is so because

as one went from ischemic to normal myocardium there was less Q and more R or QR waves, and because there was a histopathologic correlation in the infarcts examined so that the more fibrous tissue replacement the more Q wave, and the more contractile muscle the more R or QR wave (Fig 5). It is hoped that when applied as a test of benefit for surgical procedures producing auxiliary blood supplies to the heart, this electrocardiographic method will be satisfactory. Thus in a dog's beating heart, previously prepared with an efficient auxiliary collateral blood supply, these Q waves of myocardial ischemia should not be recorded over the occluded muscle mass after ligation of the anterior descending coronary artery.

SUMMARY AND CONCLUSION

1. It was stated that most surgical procedures being used in the management of coronary artery disease are designed to increase the normal, natural collateral circulation of the myocardium. Such surgical procedures are in need of objective methods of evaluation in the dog's beating heart.

2. Fluorescein injected intravenously may be used to detect changes in blood supply to the myocardium in the dog's beating heart when photographed in color under near ultraviolet light at 50 to 100 frames per second.

3. In similar slow motion movies of the dog's beating heart, alterations in myocardial contractility can be visualized after ligation of the anterior descending coronary artery. This loss of contractility is associated with ischemia of the occluded muscle mass and results in the cinematographic visualization termed "ballooning."

4. Electrocardiographic tracings of experimental coronary infarcts taken directly from the surface of the dog's beating heart showed that the "through and through" ischemic areas could be detected by the Q waves reported over them. The more ischemic the myocardium, the more Q wave, the less ischemic the myocardium, the more R wave was recorded. In intermediate areas QR waves were recorded.

5. These three visual methods utilizing fluorescein, contractility, and electrical activity of the dog's beating heart are offered as tests of benefit from surgically induced auxiliary collateral coronary circulation.

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DISCUSSION

DR OSLER ABBOTT, Atlanta.—I certainly feel that Dr Goldman is to be congratulated in presenting us with another approach in regard to experimentation, which certainly has a tremendous potential. I would like to mention briefly some work which has been carried out by the Medical School at Emory University and the Georgia Institute of Technology. They have perfected a camera system which allows an excellent visualization of the human coronaries in action by intravenous diodrast angiocardigraphy. Some of you, I am sure, viewed this film at the meeting of the Southern Medical Association last fall. This has many applications in the study of the circulation of the lung before and after operative procedures for emphysema. The major benefits are three. One is research value, for by using 16 to 64 film exposures per second and then converting it to slow motion, we can study motions too fast for the human eye. It also allows study of bronchial motility and esophageal motility too rapid for the human eye, therefore, its research potential is quite obvious. Its value as a teaching agent, to allow a permanent record of any unusual fluoroscopic occurrence in a clinical patient, is possibly its greatest contribution. Its clinical applications have been helpful to us thus far in allowing us to differentiate between infundibular stenosis and valvular stenosis in the congenital cyanotic heart group and also in other cardiac defects. We consider it as another method of improved contrast visualization which is now ready and available in the human being. Because cineangiography can be educational, and of research value, I felt it should be discussed along with the contribution of Dr Goldman.

DR ARTHUR M. VINEBERG, Montreal.—I should like to congratulate Dr Goldman on his excellent presentation.

The site at which he has tied the anterior descending branch of the left coronary artery is quite a distance from its origin. A ligature placed in this position fails to include branches which arise proximal to the ligature. When a ligature is placed at the origin of the anterior descending branch of the left coronary artery, there are very few branches that escape and, therefore, very few branches form collateral circulation. There is great variation in the published figures for survival after anterior descending branch ligation. This can be explained by failure of the ligature to include all branches.

In some animals it is impossible to obtain all the branches because occasionally branches arise directly from the trunk of the left coronary artery. Dr Goldman has demonstrated beautifully how such an artery which has escaped occlusion by the ligature can act as a collateral and revascularize the area formerly supplied by the anterior descending branch. He has further given visual evidence that any survival experiments depending on ligation of the left coronary artery must include a careful inspection of the site of ligation and of the left coronary artery for branches not included in the ligature.

DR GOLDMAN (Closing).—I am sorry we did not have time to show the technique of direct leads to explore the heart's surface. In answer to Dr Vinberg's question as to where the collateral coronary vessels are, I do not think it makes a great deal of difference if one ligates at the midportion or at the origin of the anterior descending artery, still the collateral coronary circulation would be present. It appears that it is the pressure in the two coronaries which determines whether or not the collateral circulation will be functional. If the two coronaries are occluded there will not be enough pressure to open collateral circulation. This is clinically borne out because so commonly death results when both coronary arteries are ligated in a dog or if they are occluded by arteriosclerotic processes in human beings. On the other hand, if one artery is occluded, there is good chance that the collateral coronary circulation coming from the other artery will be effective. However, this collateral circulation is not without its limits as we all know from clinical study, and even in the dog it is limited. A small occluded myocardial muscle mass produces often no visible cyanosis and no visible change with fluorescein or in contractility although there may be electrocardiographic Q or QR waves.

With the direct exploring electrode applied to the surface of the heart it appears that electrical changes which would be brought about by surgically induced coronary circulation could be readily detected. These changes of Q and QR waves should result after experimental ligation of the anterior descending coronary artery. Thus the presence or absence of Q and QR waves after experimental coronary ligation in a prepared dog's beating heart should indicate the effectiveness of the prepared additional surgically induced coronary collateral circulation.

ARTIFICIAL OXYGENATION AND CIRCULATION DURING COMPLETE BY-PASS OF THE HEART

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A KEEN interest in the problems of extracorporeal circulation has been aroused and maintained in many surgeons by the promising results of the experiments of several groups of investigators.^{1,2} This interest had, as its background, or was at least nurtured by, the signal advances made in surgery^{3,4} in the past fourteen years. In the writings of Kolff, Jongsloed, Gibbon, Miller, and Dennis, physicians have access to excellent summaries of methods of artificial oxygenation and circulation, and to interpretations of correlative, physiologic phenomena.

In this publication, the purpose is to report an experience with the dispersion oxygenator and pump described by Clark, Gollan, and Gupta.^{1,2} Their heart lung apparatus employs principles differing from those of the mechanical devices of other investigators.^{3,4} This machine depends upon dispersion for the introduction of oxygen into the blood and for the elimination of carbon dioxide. It provides for the removal of these gases, carbon dioxide, and excess oxygen, in the form of bubbles, before the blood is returned by pump into the animal. The pumping effect of this machine is obtained without the use of roller, diaphragm, or piston systems.

This report, based on the initial experience of the authors, does not deal with many of the problems emphasized by authoritative investigators. It is concerned, on the other hand, with some of the questions confronting a group of workers starting to experiment in this field.

DESCRIPTION AND OPERATION OF THE APPARATUS

Fig. 1 is a diagram of the oxygenation and pumping unit. The venous blood flows from the cannula, draining the superior and inferior vena cavae into the oxygen dispersion unit, at the base of the apparatus. (A, entrance for venous blood). The first step in oxygenation and in the elimination of carbon dioxide occurs as the blood flows upward in the inner, sintered glass channel of a double-walled Pyrex chamber. (C, double walled oxygen dispersion chamber.)

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†The three Pyrex units mentioned in the text may be obtained from the Laboratory and Pharmaceutical Sales Division, Corning Glass Works, Corning, New York, designated as Model C 8 inch dispenser, defoaming chamber and pumping chamber.

The oxygen, which is delivered at a pressure of 5 to 10 pounds per square inch into the glass jacket surrounding the central porous-walled cylinder, passes through the microscopic openings and enters the blood in the form of tiny bubbles (*B*, entrance for oxygen). The size of the bubbles is determined by the diameter of the pores (4 to 5 microns) in the wall of the central cylinder and possibly by the surface tension of the blood. The surface of the sintered glass must be wettable by blood. A hydrophobic, porous surface produces large

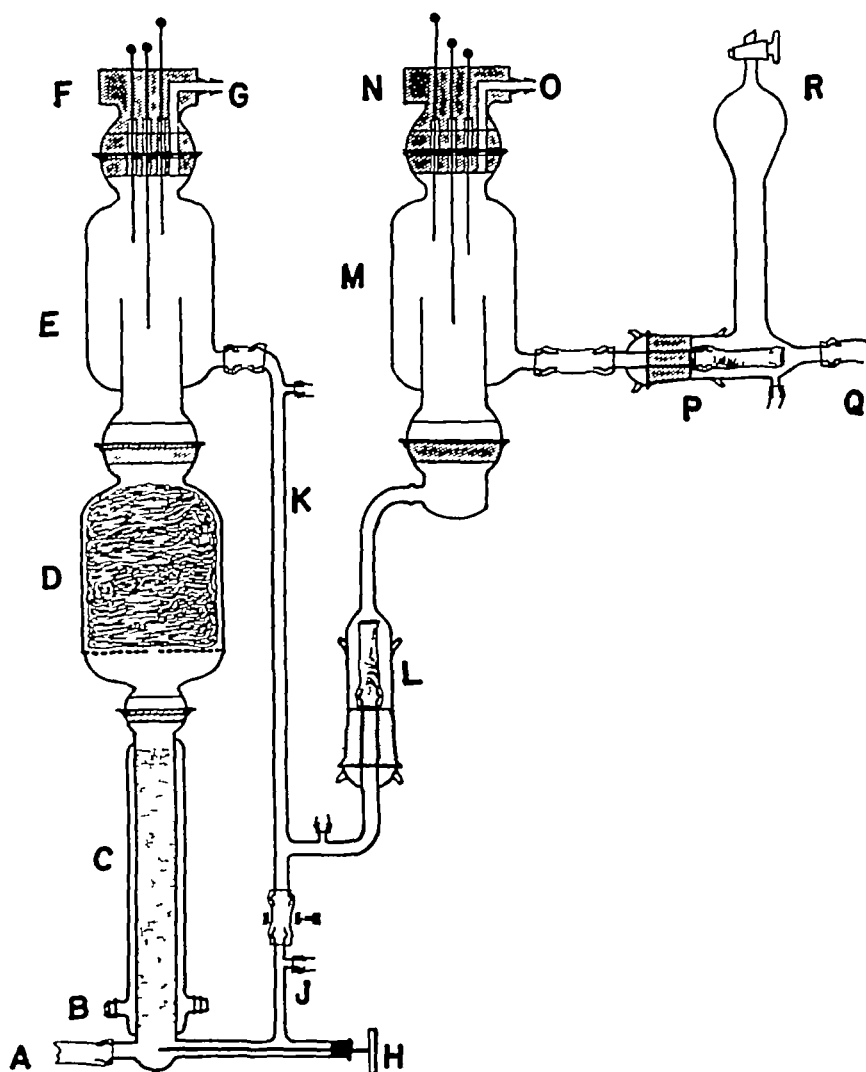


FIG. 1.—A, entrance for venous blood; B, oxygen entrance into outer jacket; C, double-walled oxygen dispersion unit; D, coil sensing chamber; E, reservoir; F, electrode holder; G, attachment for suction line; H, thermometer; I, bypass channel for venous blood; J, channel for arterialized blood; K, valve; L, pumping chamber; M, electrode holder; N, attachment for suction pressure line; O, valve; P, outlet for arterial blood; Q, clastic chamber; R, valve.

bubbles, which are less efficient than small ones in this process of oxygen and carbon dioxide exchange

The blood must be freed of these tiny bubbles (excess oxygen and carbon dioxide) before it can be pumped back into the animal's vascular system. This second step in the extracorporeal circuit is completed in the Pyrex coalescence chamber, which is sealed to the upper end of the oxygen-dispersion unit (*D*, coalescence chamber). In this chamber the fine mist of tiny bubbles coalesce to form large bubbles which are easily withdrawn from the apparatus. Bubble coalescence is obtained by passing the blood over a broad surface coated with polymethylsiloxane defoaming compound (DC Antifoam A) *. The coalescence chamber is packed with fine polyethylene fiber which is coated by 2 Gm of Antifoam A, evaporated from an ether solution. To date, experiments with this organo silicon oxide have shown it to be innocuous¹⁴

The large bubbles and the blood, the latter enriched with oxygen and freed of some of its carbon dioxide, continue their upward course from the coalescence chamber to enter the third stage of the extracorporeal circuit. The glass chamber used for this step is the third in the vertical line, and serves as a reservoir and avenue for the elimination of bubbles (*E*, reservoir). The large bubbles then leave the surface of the blood and are removed by the suction to which this chamber is exposed (*G*, attachment for suction line). The intermittent suction effect is controlled by an electrode projecting at an upper level in the chamber. A minute current flows through this electrode when the blood level rises high enough to contact it. The current causes a solenoid to interrupt the suction for as long as the electrical circuit is complete.

The fourth step in the extracorporeal course of the blood is the process of pumping it back into the vascular system of the animal. The specifications of the Pyrex pumping chamber are the same as those for the reservoir (*M*, pumping chamber). Pumping is done by the alternation of suction and pressure over the surface of the blood in this chamber (*O*, attachment for suction-pressure line). No moving parts are in contact with the blood in a pump employing this principle. This pulsating action is controlled by two electrodes supported at different levels in the upper half of the chamber. A very small current flows through each electrode when blood rises high enough to contact it. This current causes the solenoids in the suction and pressure lines to open and close at the appropriate moments. When the blood level rises high enough to touch the end of the upper electrode, the suction is interrupted and the pressure phase begins. When the blood level falls away from the tip of the lower electrode, the pressure is discontinued and suction is once more applied.

A narrow by-pass channel, with an adjustable clamp, serves to shunt venous blood from below the oxygen dispersion unit into the stream of "arterialized" blood before it enters the pumping chamber for return to the animal (*J*, by-pass channel, *K*, channel for "arterialized" blood flowing to pumping chamber). The volume of blood shunted around the first three steps of the circuit may be

*Made by Dow Corning Company, Midland, Mich.

varied by adjusting the screw clamp on the tubing in the by-pass line. The shunting of *some* venous blood into the arterialized stream serves to counterbalance any excessive oxygenation which develops in the oxygen-dispersion unit. The blood temperature is measured directly by a thermometer (*H*) which projects into the stream entering the system.

The Pyrex "elastic" chamber (*R*) buffers the pulsating stream from the pumping chamber. The elastic unit maintains some pressure effect on the arterial stream while the pumping chamber is refilling in the suction phase (*Q*, outlet for arterial blood). The flow rate is increased by this effect.

The only two valves (*P* and *L*) in the entire system are the simplest type and are made of Silastic in the shape of flat tubing.* The electrodes are stainless steel rods, which are easily adjusted up or down in their Lucite holders (*F* and *V*). Only 5 to 10 microamperes flow through these electrodes when the circuit is completed by contact with the surface of the blood. The current of 10 microamperes does not cause any damage to the blood in the circuit. Except for the porous unit for oxygen-dispersion, all the glass parts and the plastic tubing (Surgical Koroseal or Tygon S-22-1) used for making connections are coated with silicone. This is done by wetting the parts and tubing with a 3 per cent benzene solution of DC-1107,* allowing them to dry, and baking them for several hours at 150° C. The coating is reapplied after 20 experiments, or when the surfaces no longer appear hydrophobic.

A satisfactory source of suction and pressure may be found in a variety of standard pieces of laboratory equipment. (The Whirlwind machine† was used as the source of suction in these experiments.) The pressure for pumping was obtained from a tank of oxygen.

The assembled apparatus is put through a test run after all the parts have been securely joined together, during this period, the pressure on the pumping line is increased gradually to 14 pounds per square inch, a pressure which exceeds that used in arterial perfusions. The vacuum pump is adjusted so that the suction is between 6 and 10 inches of mercury. The oxygen pressure in the outer jacket of the dispersion unit is increased, until a mistlike current of bubbles is seen entering the coalescence chamber and an excess of the gas starts escaping from the surface of the liquid in the reservoir. The pumping rate is controlled by regulating the clamp on the suction-pressure line, or, if desired, the rate may be controlled by regulating a clamp on the arterial line leading from *Q* (Fig. 1).

In cleaning the apparatus saline is first circulated through it, then water, and finally water containing household "chlorine" water. The parts are completely disassembled and individually washed and dried. The polyethylene fiber in the coalescence chamber is cleaned by rinsing with water, then with 95 per cent alcohol, and finally with ether in order to remove the antifoam compound. The coalescence unit may then be allowed to dry, or the polyethylene fiber may be recoated immediately with antifoam compound. The porous glass wall in the oxygen dispersion unit requires meticulous cleaning. For a thorough

* Made by Dow Corning Company, Midland, Mich.
† Obtained from Max Wheeler and Company, Cincinnati, Ohio.

insing of the sintered surface, a stream of strong chlorine water is passed into the oxygen inlet of the outer jacket. The unit then stands overnight in concentrated sulfuric acid, to which a few crystals of sodium nitrite have been added. This acid is removed by drawing a 5 per cent solution of sodium bicarbonate through the porous wall.

The entire apparatus and its supporting frame of aluminum rods can be placed on its side in an autoclave of standard size. This method of sterilization has no adverse effect upon the plastic tubing, which is used to join the chambers and for attaching the machine to the cannulas inserted in the animal. The Silastic valves and the Lucite electrode holders are not altered by autoclaving. It is necessary, however, to sterilize the polyethylene fiber-filled coalescence chamber by soaking it in a germicidal solution.

OPERATIVE PROCEDURE

The animals used in this series were all mongrel dogs between 1 and 3 years of age, varying in weight between 30 and 35 pounds. Each animal was observed for 2 to 4 days preoperatively, and an estimate of cerebral function and normal activity was made for comparison with the condition following total by-pass of the heart and lungs.

Although the apparatus used fits into a standard autoclave, no attempt was made in any of the experiments to adhere to aseptic surgical procedure. The animals were shaved and washed prior to surgery, in order to eliminate as much gross contamination as possible, but none of the drapes, instruments, and equipment were sterilized.

The animal selected for pumping was premedicated with morphine and atropine prior to the intravenous administration of Pentobarbital Sodium. Large operative wounds for the exposure of the heart and great vessels in animals whose blood is rendered incoagulable by heparin, as in these experiments, necessitated meticulous attention to possible sources of bleeding during the operative procedure. No tissue encountered in the dissection was divided without first being clamped. The small portions of tissue on each side of the proposed line of division were divided, and each of these bundles was individually ligated. The right pleural cavity was entered through the fourth intercostal space, and the femoral arteries, femoral veins and the right common carotid were dissected free for a distance long enough to permit cannulation.

When the chest of the animal was opened, controlled pressure was applied to the breathing bag attached to the endotracheal tube. The rib margins were separated with a self-retaining retractor, and the right lung was displaced so as to expose the superior vena cava. The azygos vein was divided and the pericardium was opened, in order to expose the right auricle and the inferior vena cava. A rubber ribbon was passed around the superior and inferior vena cavae, at their entrance into the right auricle. A measured dose of heparin was then given intravenously, and the peripheral cannulations carried out. When the animal was ready for total by-pass of the heart and lungs, the two rubber ribbons around the superior and inferior vena cavae were tightened around the limbs of

the right heart cannula. This tightening effectively prevented the return of venous blood to the right heart from the peripheral circulation. During the period of total by-pass, an incision was made in the right auricular appendage to permit exploration, under direct vision, of the interior of the right auricle. The venous return in the coronary sinus was drawn off by a simple suction device and was then measured. (The presence of air in the venous pick up line of a dispersion-oxygenator is of no consequence, therefore, this blood can be returned to the venous side of the apparatus.)

Anticoagulants—In the first eight experiments, the donor blood, for filling the machine and for replacing blood lost from the incisions in the recipient animal, was collected in standard transfusion bottles containing 120 cc of ACD solution. When this blood was pumped into recipient dogs there was a rapid and irreversible fall in blood pressure. Donor blood was therefore collected, with heparin as the anticoagulant (50 mg per 1500 cc of blood).

In the first 33 experiments, the recipient animals were given heparin in dosages of 3 to 4 mg per kilogram of body weight, just prior to the insertion of the cannulas. This dosage was adequate for maintaining an incoagulable state during the pumping period. However, near the end of the pumping period, small collections of material thought to be fibrin were noted on the walls of the pumping chamber. The heparin dosage was revised upward until the formation of this material ceased. Prior to cannulation, all the dogs in the remainder of the series received 10 mg of heparin per kilogram of body weight.

Reversal of the heparin effect at the conclusion of circulation through the experimental circuit remained an unsolved problem. Many of the first animals received protamine sulfate in doses of the number of milligrams equal to the amount of heparin injected. Some of these animals suffered severe hypotension on administration of protamine, even when this was given very slowly and in high dilution. Transfusion of fresh, whole blood, collected in a syneon syringe from a donor dog, seemed to be more effective in reversing the heparin effect. This approach to the clotting problem, although not ideal, proved effective enough to permit eliminating the use of protamine in many dogs later in the series.

Eight animals in the entire series died of postoperative hemothorax, and fatal bleeding seemed to have come from the region of dissection in the mediastinum. This bleeding developed in spite of the meticulous ligation of all bundles as the tissues were divided. However, only two of the final 23 dogs used in the series died of uncontrolled oozing. This reduction of mortality from postoperative hemorrhage was attributed to two factors—the liberal use of fresh, whole blood and the use of the multiple ligation technique.

Cannulation—An L-shaped polyethylene tube, similar to the cannula described by Leeds,¹² was used in the first 42 experiments for the collection of the venous blood returning to the right auricle from the superior and inferior vena cava. This tube was inserted into the superior vena cava through the proximal end of the divided azigos vein, and down into the inferior vena cava through the right auricle. The stump of the azigos vein was then ligated about

the stem of the cannula. In the course of these 42 experiments, three serious faults in this system were observed. First, the venous return was often inadequate to maintain a satisfactory flow rate. This occurred because of a tendency for the walls of the superior and inferior vena cavae to pull into the openings of the cannula during the suction period of the pumping cycle, and to effectively block the cannula. Second, the insertion of such a cannula was attended by the risk of introducing air into the right auricle. Third, the upright limb of the cannula blocked direct operative approach to the heart.

The problem of venous return through a cannula inserted into the superior and inferior vena cavae through the right auricle was managed at first by the incorporation of a suction-release mechanism. Through this device, the suction was automatically turned off when the indrawn walls of the superior and inferior vena cavae blocked the cannula. Flow rates were increased by this mechanism, but were still not the best obtainable.

Rapid, continuous flow of venous blood into the machine was eventually obtained by the use of a "femoral vein supplement." This consisted of a long polyethylene catheter with multiple perforations in its wall. The catheter was passed into the inferior vena cava through a femoral vein, and was threaded through the intracardiac portion of the L cannula into the superior vena cava. The end of the "femoral vein supplement" catheter, which remained protruding from the femoral vein, was sealed to prevent blood from escaping and air from entering. The small, indwelling, perforated catheter made it mechanically impossible for the walls of the superior and inferior vena cavae to collapse completely. Through this device there was uninterrupted filling of the machine, and flow rates between 2,500 and 3,000 cc per minute were easily obtained.

The danger of air embolism during the insertion of the cannula was eliminated by the use of a Cellophane dialysis-tubing obturator in the lumen of the cannula. A length of Cellophane tubing was closed at one end, and its outer surface was lubricated with a small amount of mineral oil in order to facilitate removal. It was then inserted into the L cannula and distended with saline. The Cellophane sausage thus formed occluded the lumen completely, and permitted insertion of the right heart cannula without danger of air embolism or hemorrhage. When the cannula was in place, the oiled obturator was removed, and its pistonlike action on removal filled the cannula with blood.

At the termination of total by-pass, closure of the heart and removal of the right heart cannula were accompanied by the danger of air embolism. The possibility of trapping air in the opened heart was reduced by allowing the heart to fill completely, before closure, with blood from the coronary sinus,⁴ or by pouring in saline to fill the auricle. The danger of air embolism during the removal of the L cannula was minimized by filling the pleural space with saline, withdrawing the instrument, and clamping the vein under the level of this solution.¹⁶

Several methods for the return of oxygenated blood to the animal were evaluated. In early experiments, oxygenated blood was returned through both femoral arteries in a retrograde fashion by the use of polyethylene catheters

passed into the arch of the aorta. Arterial return by this method was adequate, but high pumping pressures were necessary in order to overcome the resistance in long catheters. Return of oxygenated blood was attempted through one femoral artery catheter and one common carotid artery cannulated with a T tube, so that both limbs of the divided carotid artery functioned as arterial in

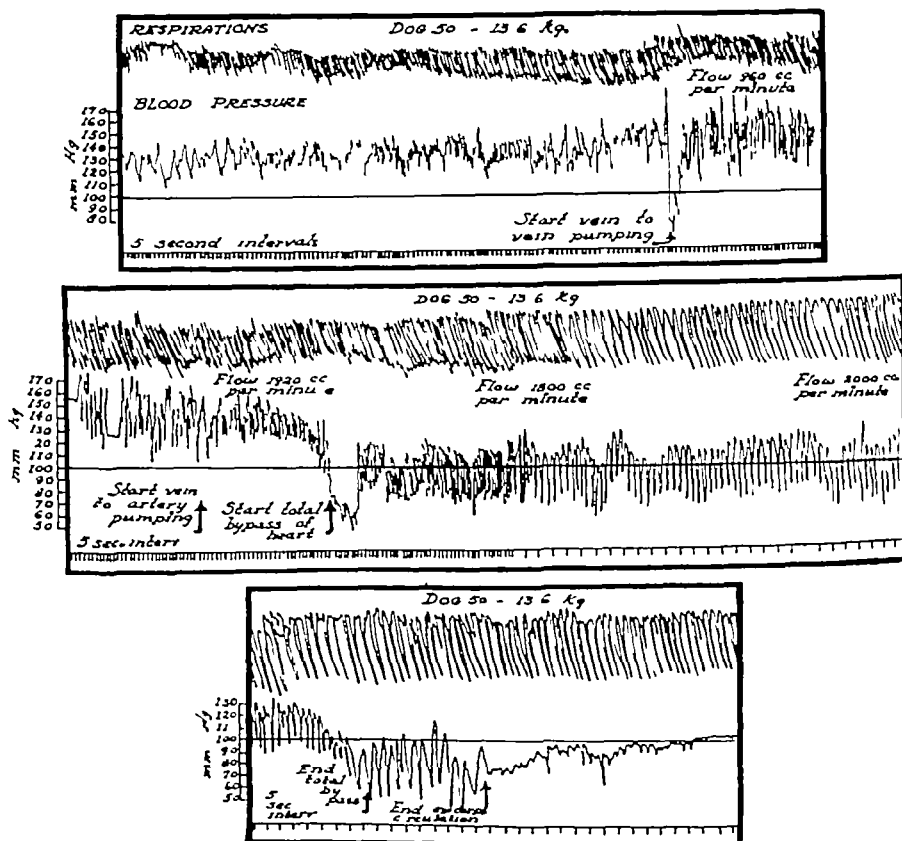


Fig. 1.—Portions of records of Dog 50. Tracing of respirations and blood pressure with flow rate and changes in extracorporeal circuit.

lets. This was discontinued because orbital edema on the same side seemed to result. In the most recent 20 experiments the arterial return was carried through two polyethylene cannulas. One was pointed centrally in the right femoral artery and the other was pointed toward the heart in the right common carotid artery. These cannulas were 4 mm. in diameter, and were inserted for only short distances.

Two additional cannulations completed the pick up and return systems. A catheter was placed in the right femoral vein and this served as a channel for return during vein to vein pumping. In order to record blood pressure and to obtain samples for oximetry the left femoral artery was cannulated.

By-Pass of the Heart—After the right heart cannula was connected to the venous pick-up tube and the arterial inlet connections were made, the pump was turned on. For the first few minutes the blood was drawn out through the right heart cannula oxygenated and returned through one remoral vein. (This was the period of vein-to-vein pumping indicated on Figs. 2 and 3.)

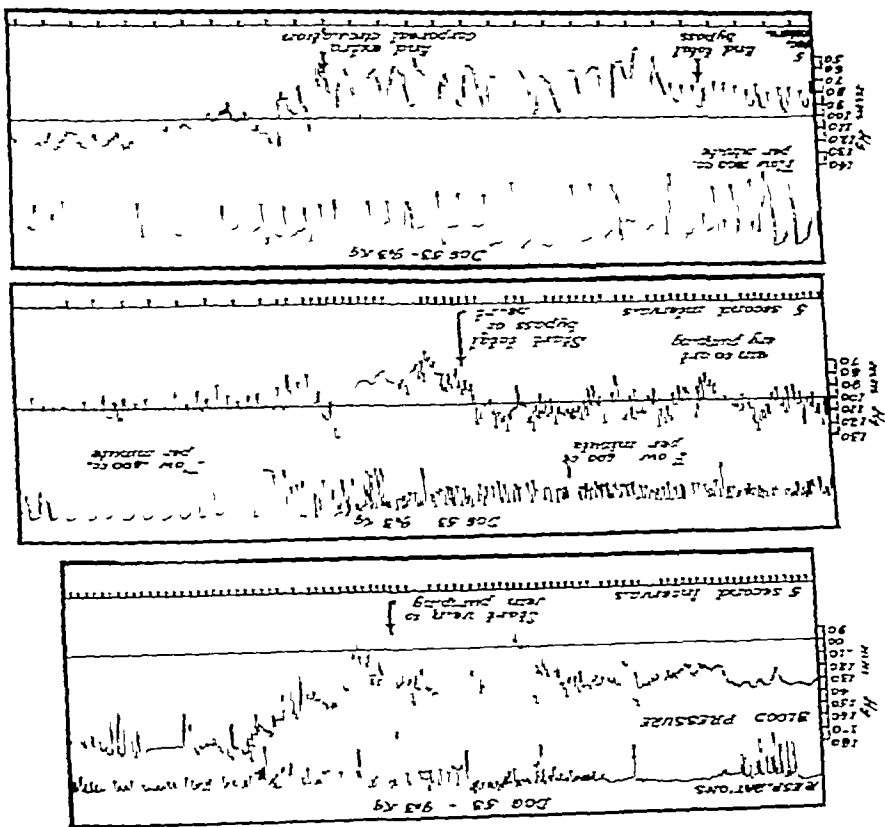


Fig. 3—Portions of record of Dog 53. Tracing of respirations and blood pressure with flow rate and changes in extracorporeal circuit.

When a satisfactory level of oxygenation was established, the remoral vein was closed and the cannulas in the remoral and carotid artery were opened. This vein-to-artery, partial by-pass was continued for a short period, during which time mechanical adjustments were made. The two rubber ribbons about the superior and inferior vena cava were then tightened so as to compress the caval pass around the stem of the right auricle cannula. This established the total by-pass phase of the experiment, and from this time on until the end of the by-pass, the apparatus functioned with very few further adjustments.

OBSERVATIONS AND LABORATORY DATA

Continuous recording of the blood pressure respirations electrocardiogram, body temperature and urine formation was attempted in most of the experi-

ments For the most part, it is practicable here to refer only to the observations recorded in that phase of the experiment when the animal was on total by-pass of the heart Portions of the records of two experiments are shown in Figs 2 and 3 The following laboratory data cited, however, cover the full duration of each experiment and not just the crucial 30-minute period of total cardiac exclusion

The mean blood pressure remained within a normal range in most of the animals during total by-pass With regard to the breathing, it is impossible to say more than that the animals continued with normal respiratory movements, even when, in total by-pass, the lungs were deflated and pale, and practically bloodless After all the blood returning through the superior and inferior vena cavae had been rerouted through the apparatus, the volume of the heart decreased immediately and the auricles were quite collapsed During this same period

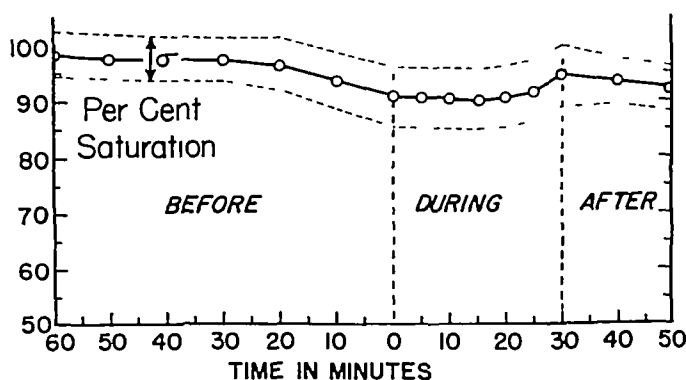


Fig 4—Oxygen saturation of arterial samples during total by-pass The open circles represent the mean values obtained Each of these averages is derived from twelve to eighteen analyses in the early period before pumping In the period immediately before during and after the by-pass between 25 and 35 analyses were averaged for each point In the late period after by-pass between eight and thirteen analyses were averaged for each point Each figure records the analyses of a total of 325 arterial samples The standard deviation from the mean is indicated by the dotted lines and is obtained by the following formula
$$s = \sqrt{\left(\frac{1}{N}\right) (\sum x^2) - M^2}$$

the ventricular contractions remained strong and tended to slow down There was no gross disturbance in the electrocardiogram, and in a few experiments, the animal had an occasional ventricular extrasystole With the wall of the right auricle opened wide, the return from the coronary veins, collected by direct aspiration of the coronary sinus in several animals, measured 30 to 40 cc per minute The body temperature was allowed to fall to subnormal levels (32° to 36° C) throughout many of the experiments In the latest group, the body temperature was held at a nearly normal level (36° to 38° C) by adding a heating coil to the oxygenating unit and by decreasing radiant heat loss from the animal The formation of urine did not seem to be interrupted in the total by-pass phase of the experiment The survivors often regained consciousness at the time of closure of the chest wall incision, and within four hours after completion of the experiment they stood on all fours and started to drink water

The oxygen saturation of arterial samples ranged between 93 and 98 per cent in the control period before pumping (Fig 4). During the total by-pass or heart and lungs, the oxygen saturation averaged between 90 and 95 per cent. After restoration of normal circulation, oxygen saturation in arterial samples varied between averages of 90 and 93 per cent. At no time in the interval of total by-pass did the averages of arterial saturation fall below 90 per cent.

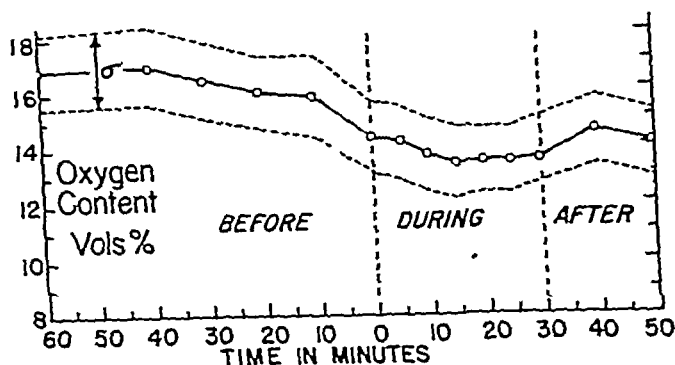


Fig 5—Oxygen content of arterial samples during total by-pass. (For further explanation see Fig 4.)

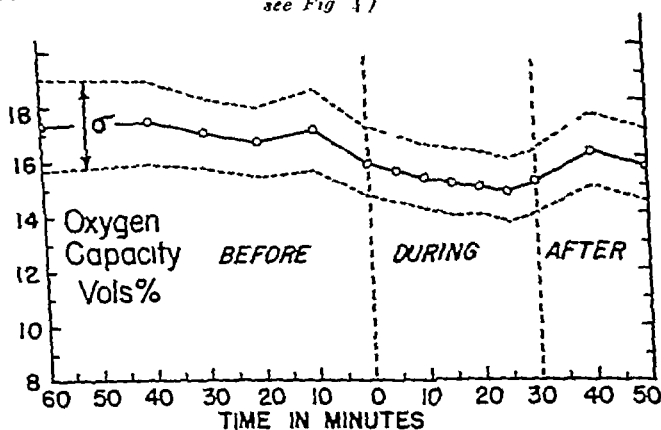


Fig 6—Oxygen capacity of arterial samples during total by-pass. (For further explanation see Fig 4.)

The oxygen content of the arterial blood shown graphically in Fig 5 was reduced slightly during the by-pass period. This change was due in part to the addition of fluids not containing hemoglobin (physiologic saline solution, 5 per cent glucose in water, etc.) That this was a factor was indicated by the fall in oxygen capacity which occurred at the same time (Fig 6). Both oxygen content and capacity rose somewhat after the conclusion of by-pass, probably as the result of transfusions of whole blood.

Prior to the period of artificial oxygenation, arterial samples varied considerably in their total carbon dioxide content, as shown in Fig 7. This probably resulted from variations in the method of anesthetizing and of administering oxygen. The total carbon dioxide content was increased in arterial samples

taken during the by-pass period. After the pumping, the trend of the total bicarbonate was downward and approaching control levels. A carbon dioxide absorbing unit was inserted in the line (Fig 1, G), through which all of the carbon dioxide and excess oxygen were withdrawn from the system. By this absorption unit it was possible to use a gravimetric method for measuring the amount of carbon dioxide eliminated in the process of dispersion oxygenation. The average in six experiments was 40.3 cc per minute during the period of total by-pass.

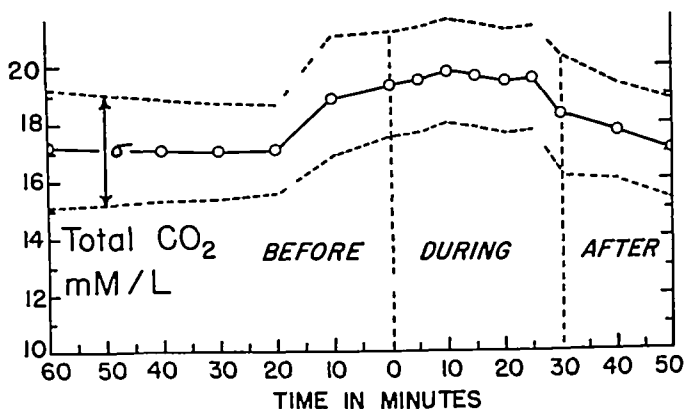


Fig 7—Total carbon dioxide content of arterial samples during total by pass. (For further explanation see Fig 4)

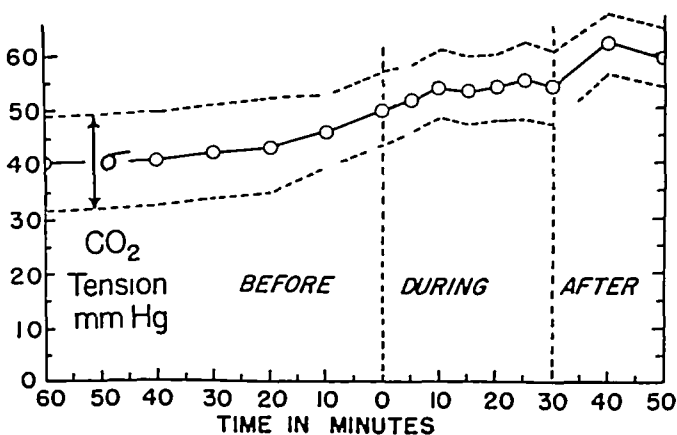


Fig 8—Carbon dioxide tension of arterial samples during total by pass. (For further explanation see Fig 4)

Analysis of arterial blood drawn before extracorporeal circulation was started showed a tendency for the carbon dioxide tension to rise (Fig 8). This tendency was accentuated after pumping was started, with the values remaining at about 54 mm Hg during the remainder of the period of complete by-pass. Following the period of by-pass, the carbon dioxide tension rose somewhat higher

During the prepumping period of the experiments the plasma pH gradually fell, as shown in Fig 9. This change was probably due to retention of carbon dioxide, resulting from the method of anesthetization and inflation of the lungs. During the by-pass period, the tendency toward acidosis was somewhat aggravated. In the postpumping period the pH fell further.

The criteria of survival were based on observations made in the period immediately following total by-pass. No animal was considered a survivor unless it made a complete recovery from the anesthetic, evidenced good cerebral function, walked, ate, drank, voided, and defecated. Liberal doses of antibiotics were administered, but long-term survivors were few. Only seven of these animals lived for several months before they were sacrificed, the others that had met the criteria of survival succumbed to intrathoracic and generalized infection or to thrombosis of the superior vena cava, in a period of forty-eight hours to two weeks (See Table I).

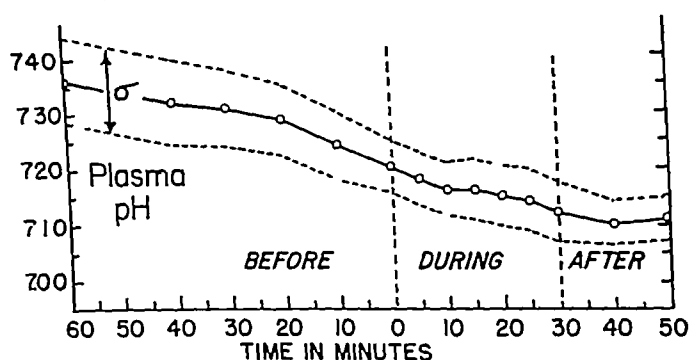


Fig 9—The pH of arterial samples during total by-pass (For further explanation, see Fig 4)

TABLE I ANIMALS THAT MET CRITERIA OF SURVIVAL

DOG NO	DATE OF BY-PASS	DATE OF DEATH	REMARKS
8	5/26/51	8/ 3/51	Sacrificed Normal autopsy
12	7/ 7/51	9/17/51	Sacrificed Dense right sided pleural adhesions
16	7/26/51	9/ 9/51	Sacrificed. Pericardial effusion and subcutaneous abscesses in chest wall
17	7/27/51	9/ 9/51	Sacrificed. Normal autopsy
21	9/ 1/51	10/15/51	Died. Chylothorax and compression atelectasis
37	11/10/51	-----	Living
38	11/11/51	11/14/51	Died Marked pulmonary consolidation
41	11/24/51	11/26/51	Died Thrombosis of the superior vena cava
45	12/22/51	12/24/51	Died Marked pulmonary consolidation and edema
47	12/29/51	1/11/52	Accidentally electrocuted
50	1/ 6/52	-----	Living
51	1/12/52	1/13/52	Died Pulmonary consolidation and edema
52	1/13/52	1/14/52	Died Thrombosis of the superior vena cava
53	1/19/52	2/ 2/52	Died Multiple abscesses and purulent pericarditis
55	1/26/52	2/ 3/52	Died Thrombosis of the superior vena cava
56	1/27/52	1/29/52	Died Marked pulmonary consolidation and edema

Of the 57 dogs subjected to complete by-pass of the heart and lungs for periods of thirty to forty minutes, 52 remained alive until the extracorporeal circulation was completed. The first animal to meet the criteria of survival was the one studied in the eighth experiment. Consecutive surviving animals were obtained after 50 experiments had been completed. Sixteen dogs made complete recoveries, giving an over-all survival rate of 28.7 per cent. The initial survival rate with citiated blood for transfusion was 12 per cent on the first eight animals. The rate of survival rose to 33.3 per cent after the citrate solution in the transfusion blood was replaced by heparin as an anticoagulant. After a careful technique was instituted of crossmatching donor and recipient animals, the survival rate rose to 40 per cent. The introduction into the apparatus of a by-pass channel, which shunts a fraction of the venous blood directly into the chamber pumping out the arterial return, resulted in further improvement in the survival rate, 57 per cent of the last fourteen animals survived.

COMMENTS

From this experience with extracorporeal oxygenation and pumping of the blood, there are some observations which merit further comment. These observations are related, it is believed, to factors determining the success of this apparatus and method, and are therefore discussed in the following text.

Crossmatching Donor and Recipient Dogs—There is little doubt that the transfusion of incompatible blood caused the death of some of the animals early in the experiment. The first 29 experiments were carried out without regard for the possibility of blood incompatibility among dogs. After this, using the direct slide method, the recipient's cells were always matched with plasma from the prospective donors.¹⁷ (Approximately 25 per cent of the tests showed strong agglutination.) Ancillary experiments were carried out in which heparinized blood known to be incompatible was given as replacement transfusions to two dogs. The first animal became hypotensive and died within twenty minutes. The second remained anuric and died after twenty-four hours. The autopsy findings in both animals were renal ischemia and infarction, and severe pulmonary edema.

Carbon Dioxide Elimination—It has been suggested that the reduction of serum p_H and elevation of carbon dioxide tension may have harmful effects on dogs subjected to total by-pass of the heart and lungs.¹⁸ The possible significance of these changes was considered in the analysis of the early experiments in which the dogs succumbed. Collateral studies were made with three dogs re-breathing carbon dioxide in an oxygen-filled bag. The p_H of their arterial blood was followed by frequent determinations with a Beckman p_H meter. Ten minutes after the start of re-breathing the p_H fell from normal to 7.2. After eighteen minutes the p_H was 7.11. For thirty minutes, the animals remained with an arterial p_H of 6.90 to 7.13. Following this period they recovered from their anesthetic without delay, and were normal in all respects. It is known that in arterial p_H, if severely reduced for two to three hours, may cause death in dogs.¹⁹ However, the severe acidosis, if present for relatively short periods, may not have a harmful effect.

Filters in the Extracorporeal Circuit—Fine mesh, stainless steel and sintered glass filters were employed in some of the experiments. These porous surfaces caught some small clumps of erythrocytes and fibrin masses, but it was difficult to ascertain that the filtering surface was not a factor in the formation of these tiny, unshaped groups. With the increased dose of heparin employed in the most recent experiments and with the crossmatching technique, need for a filter was probably reduced. All except three of the surviving animals were maintained without a filter in their extracorporeal circuit.

Thrombosis of the Superior Vena Cava—Thrombosis of the superior vena cava was the cause of death in three dogs that had made complete recovery from the period of total by-pass. The following are the factors responsible for these thromboses, which were all at or above the level of the azygos vein and never in the inferior vena cava: first, trauma to the intima of the superior vena cava during cannulation, and while the walls of the vena cavae were pressed to the cannula in the period of total by-pass, second, the intravenous administration of protamine sulfate into the cephalic vein, relatively close to the site of caval trauma and the stump of the azygos vein, third, local bacterial infection in animals, with septicemia and abrasions of the intima of the superior vena cava.

With some practice, the cannula can be inserted without intimal trauma. Narrow rubber ribbons have been employed as ligatures, to compress the caval walls around the instrument for the period of total venous clamping. Tight ligation with a cord, with its resultant ischemia of the vessel wall, is avoided as much as possible.

Autopsy Findings on Dogs That Succumbed—A study of the results of necropsy on animals that survived for only a few hours after the total by-pass, did not always reveal the cause of death. The brain was not examined in most instances, and no gross abnormality was identified in the brains that were removed. It was noted, however, that a large proportion of the dying animals exhibited signs usually associated with cerebral anoxia in man.

A variable amount of pulmonary interstitial and peribronchial bleeding was a frequent finding. This gave the appearance of a red consolidation of the lung (Microscopic sections disclosed hemorrhage and edema.) The application of excessive pressure in a closed system for the administration of oxygen into the trachea during stages of the operation, is possibly one factor in the pathogenesis of this hemorrhage. A second factor may be the accidental needle puncture of the lung in a heparinized animal during thoracenteses carried out in the post-operative period.

In the gross, the kidneys were usually normal in size and color, with well differentiated cortical and medullary portions. On the surface of many of the kidneys examined, however, there were one or more small, cortical infarcts. These ranged from 2 to 5 mm in diameter. The microscopic sections revealed infarcted tissue and degeneration of all cellular elements.

SUMMARY

1. A description is given of a simple, all glass dispersion oxygenator with some of its effects on the blood flowing through the extracorporeal circuit.

2 The operative procedure for the experiments, the use of anticoagulants, and special methods of cannulation are given in detail

3 Observations made during by-pass of the heart and lungs are listed analysis of arterial samples for the oxygen and carbon dioxide content and pH is also included

4 Experiments on 57 dogs are described, in which there was total by pass for not less than 30 minutes (Fifty-two of these animals remained alive for the duration of the experiment) Survival with complete recovery from the anesthetic is emphasized, and 16 animals in this category are cited

5 Several specific problems associated with extracorporeal oxygenation and pumping of blood are discussed briefly

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(For discussion, see page 151)

SOME PHYSIOLOGIC ASPECTS OF THE ARTIFICIAL HEART PROBLEM

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ROBERT GERISCH, M.D. (BY INVITATION)

DETROIT, MICH

SEVERAL investigators have reported on various types of artificial hearts, to by pass the heart, either partially or totally. These investigations have been along two lines: first, total by-passing of the entire heart and lungs such as reported by Gibbon and associates¹ or Dennis and associates,² second, the unilateral substitution for one or the other sides of the heart while the other side and the lungs continue to function. Leeds, Puciss, and Siegel³ as well as Sewell and Glenn⁴ have reported substitutions for the right side of the heart. Sirak, Edison, and Zollinger⁵ as well as Kantiowitz and Kantiowitz⁶ have reported substitutions for the left side of the heart.

There have been few artificial mechanisms reported which would perform any or all of these functions. Wesolowski and Welehr have reported an artificial mechanism whereby either side of the heart or both sides could be by-passed using the lungs as the oxygenator. It is not the purpose of this manuscript to review all the various artificial mechanisms that have been reported to date. We wish to present, however, some physiologic data which probably pertain to all types of artificial mechanisms.

Analyses of several types of pumps that have given some promise of being satisfactory for maintaining the systemic circulation were made at the beginning of our investigation. The Dale-Schuster pump was selected for further analysis and testing because of certain inherent features. This type is a variable displacement pump, that is, each stroke of the pump is accompanied by discharge of a quantity of fluid governed by the actuating pressure and resistance to fluid flow in the circulatory system.

Pumping action in the Dale-Schuster pump is obtained by creating a volume change in a latex rubber finger cot which is enclosed in a cylinder (Fig. 1). The finger cot also separates the actuating air from the fluid being pumped. Volume change of the finger cot is achieved by collapsing it with negative air pressure and then expanding it with positive air pressure. Collapse of the finger cot results in liquid being drawn into the cylinder through the inlet valve located in the valve body. Expansion of the finger cot forces the liquid out of the cylinder through the outlet valve. The inlet and outlet valves are closed by pressure to provide unidirectional flow of the blood.

¹The artificial heart mechanism reported here was made for us by the Research Laboratories Division, General Motors Corporation. The viscosity studies were also made by them.

²Gift by grant from The Michigan Heart Association.

³From the Research Division of Harper Hospital.

⁴Report at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery, Dallas, Texas, May 3-11, 1952.

Positive and negative an pressures are admitted alternately to the finger cot by an an valve which can be operated at different speeds. An pressure control is obtained by use of a pressure regulator and an an-bleed valve. The maximum pressure that can be applied to the blood is the actuating pressure, providing the valves are properly designed to prevent high localized pressures at the seats when they close.

After extensive tests conducted with water to determine operating characteristics of this single pump unit, tests were run to determine the amount of hemolysis caused by the pump. No significant hemolysis of the blood occurred over comparatively short periods of emulsion.

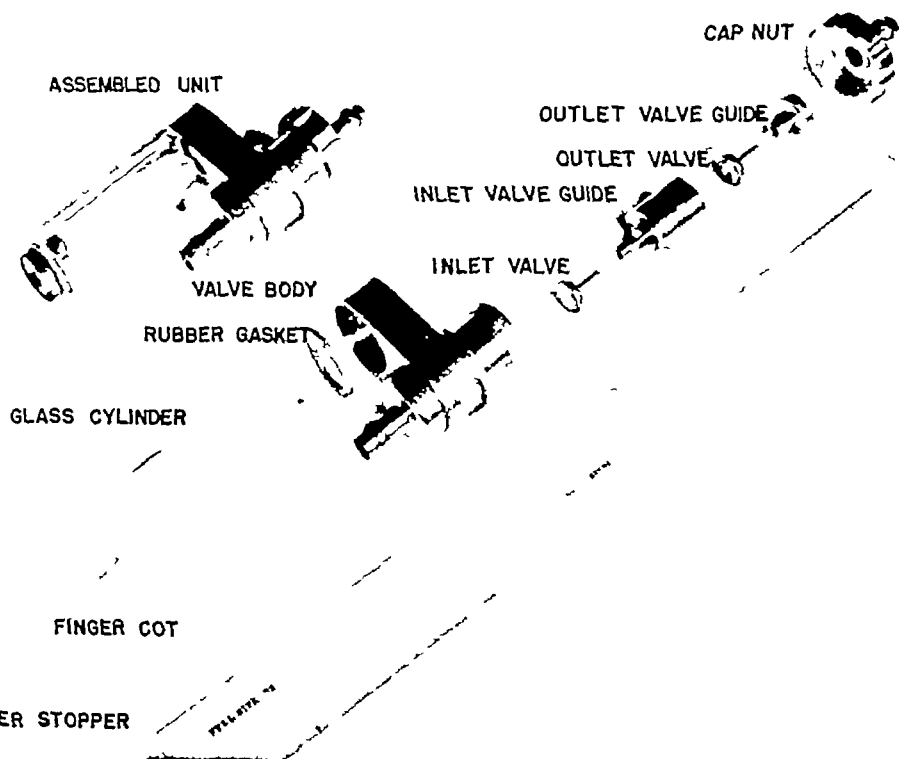


Fig 1—Individual pumping cylinder

Since our tests on this single unit gave such good results, our next step was to design a unit with sufficient capacity to accommodate an average-size adult and enough flexibility to fit the proposed medical research program. The proposed program to be followed in application of the artificial heart consisted of four steps: (1) substitution for the right heart, (2) substitution for the left heart, (3) substitution for both sides of the heart still using the lungs, and (4) substitution for both sides of the heart using an artificial lung. In other words, the mechanical heart should have two independent pumps, one to take the place of the right heart and the other to take the place of the left heart.

The peak pressure required to pump a given quantity of fluid under continuous flow conditions is less than the peak pressure required to pump the same given quantity of fluid under pulsating flow conditions. In order to minimize the blood pressure delivered to the lungs, a continuous flow pump was designed for the right side (Fig 2). To achieve a higher blood pressure in the arterial system, the left side of the mechanical heart was designed to a pulsatile flow. Sufficient pump capacity to handle a normal adult was obtained

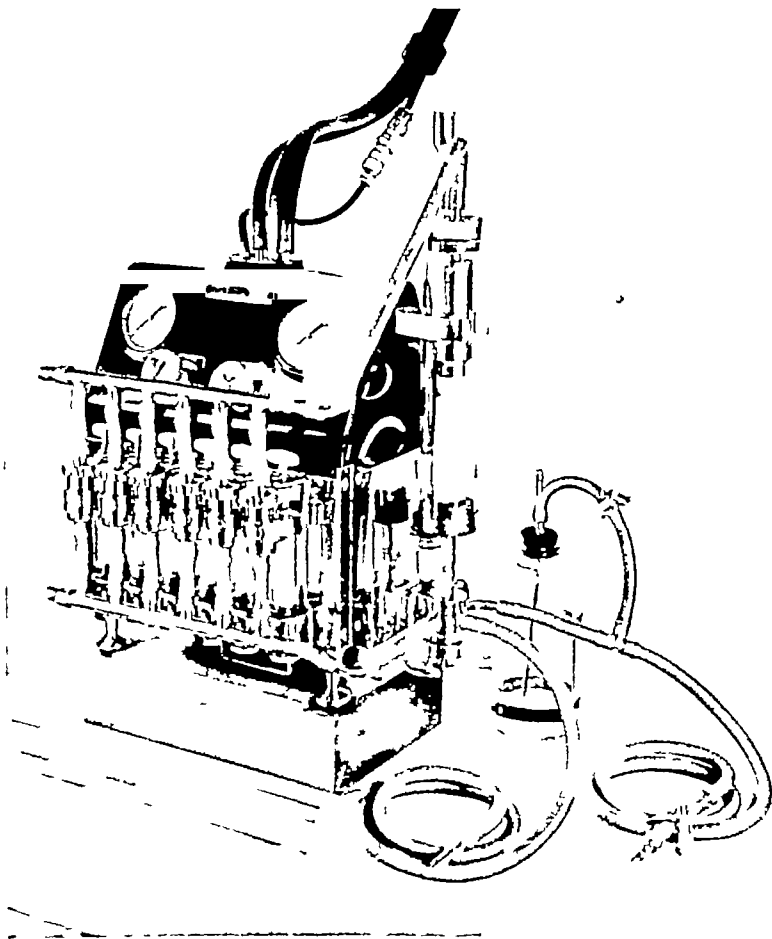


Fig. 2.—Right side of assembled pump

by using six individual units operating in parallel on each side. Constant flow on the right side was obtained by valving the admittance of air pressure to the inside of the finger cots in such a manner that one of the six individual pumps discharged every 60 degrees of camshaft rotation. The camshaft is common to both sides and it operates lever arms which valve the air pressure to the different pumps. The cams on this shaft are designed to admit positive and negative

pressures for equal periods of time to the finger cots. In order to add more flexibility to the mechanical heart, three camshaft speeds of 65, 75, and 85 revolutions per minute were made available by use of a step pulley arrangement. Separate controls for positive and negative air pressures are provided for each side.

Any number of units from one to six, can be used on either side. The number used is governed by the required capacity. Our experience indicates that three units have more than ample capacity for animals weighing 20 kilo-

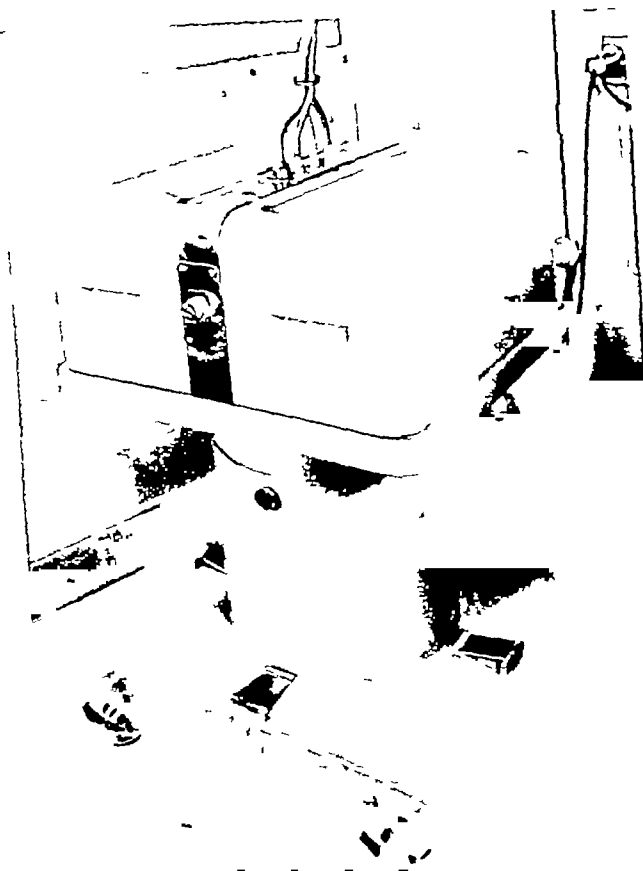


Fig 3 —Power supply for mechanical heart.

grams when using 8 mm. tubing for connections to the cannulae. Use of 12 mm tubing with six units will give the pump a capacity of approximately 5 liters per minute.

Pump parts are made of stainless steel, glass, and rubber. Standard Pyrex brand glass is used for the glass parts while the finger cots are made out of surgical latex rubber.

Individual units are very easy to disassemble for cleaning and autoclaving. All surfaces that come in direct contact with the blood stream are coated with

Dow Corning DC-1107 Silicone dissolved in carbon tetrachloride. A 2 per cent by volume solution of DC-1107 Silicone in carbon tetrachloride is used. After coating, the metal and glass parts are baked for 1 hour at 300° F. Since the Silicone coating makes the surfaces practically nonwettable, it is felt that this helps to prevent the formation of fibrin as the blood passes through the pump.

In order to measure the blood flow during operations, a Wen type flow meter was made (Fig 2). The flow rate in this meter is a function of the blood level as it discharges through a vertical slot in the upper portion of the flow meter. The slot has been calibrated to allow the flow rate values to be read during an operation. The blood discharge through the slot must occur in air or oxygen if the flow meter is to function. The amount of air or oxygen trapped in the flow meter is controlled by an air bleed valve on top of the flow meter. A filter holder is built into the flow-meter assembly. These features allow the flow-meter assembly to serve three useful purposes: flow-rate indication, filtration of the blood, and collection of any air that may enter the system. When the flow meter is used on the delivery side of either pump, a considerable pulse dampening takes place because of the large volume of air or oxygen trapped in it.

Air, pressure, vacuum, and electricity are supplied to the pump by rubber hose and electrical cable from the power supply which is located outside of the operating room (Fig 3). This power source, with the present tubing, can be located 50 feet from the operating table, although with longer tubing, it could be located a greater distance away. The pressure and vacuum are supplied by two air pumps located in this housing. One electrical switch controls the pressure vacuum pump motors and the motor located on the heart pump.

An explosion-proof motor is located on the heart pump to minimize the danger of an explosion in the operating room. The individual electrical components are all grounded through a common ground lead for safety reasons. Since air, pressure and vacuum are supplied by this power source, the heart pump is portable and can be used anywhere that regular 110 volt alternating current is available.

STERILIZATION OF THE ARTIFICIAL MECHANISM

Our mechanical heart is so devised that all parts that come in contact with the blood are sterilized by ordinary autoclaving. We believe that this is necessary in order to obtain a high degree of successful experiments. It is significant, we believe, that in our entire group of 65 experiments, not one animal has died of infection.

HEPARINIZATION

During the use of the artificial heart mechanism blood is rendered noncoagulable by using heparin solution. During the early part of our work we used too small amounts of heparin and occasionally the blood clotted in the pump. On the other hand when more than 5 mg. of heparin per kilogram of body weight was used some instances of uncontrollable oozing took place. We then began to calculate the amount of heparin to use on the basis of adding the pump volume, which in our case is approximately 300 cc. to the weight of the

animal. In other words we combine the weight of the animal with the amount of blood contained in the pump. We then put in the citrated blood which is to fill the pump, 1 mg of heparin per 100 cc of citrated blood. The remainder of the calculated dose of heparin is given to the animal. Since we have used this amount of heparin, there have been no instances of clotting in the pump and no instances of uncontrollable hemorrhage.

EFFECT OF CITRATE SOLUTION ON BLOOD VISCOSITY

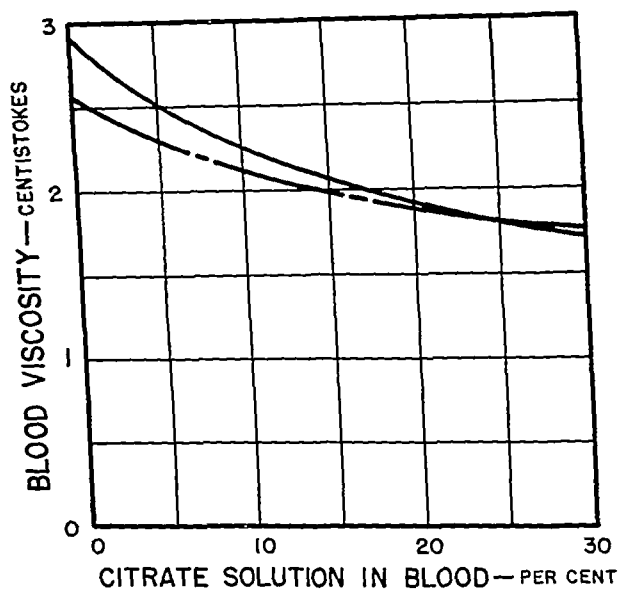


Fig 4

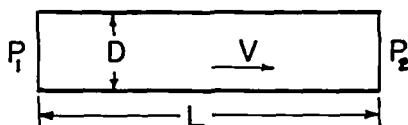
VISCOSITY STUDIES

Since it is necessary to render the blood incoagulable during mechanical pumping of the blood, viscosity studies have been carried out with anticoagulant drugs. Other investigators have found that the heparinization of blood does not affect its viscosity. We have made similar studies using the standard Ostwald Viscosimeter. Our results were the same, namely, that no appreciable change in viscosity takes place with the amount of heparin which is necessary to prevent clotting.

In addition, we have determined viscosity of blood following the administration of large amounts of transfused citrated blood. Since the viscosity of citrated solution is much less than that of blood, it is to be expected that the transfusion of large quantities of citrated blood would affect the viscosity of the circulating blood. Fig 4 shows a graph of the lowering of the viscosity of blood with given amounts of citrate solution. It is seen from the graph that

a 30 per cent dilution of blood with citrate solution produces a profound lowering of the viscosity. The two lines on the graph represent blood from two different dogs. We have also determined the viscosity of blood from dogs before and after operation of the artificial heart during which time a large amount of citrated blood was transfused. In most of these experiments there has been a profound drop in the viscosity of blood following the procedure. Although the lowering of viscosity is somewhat directly proportional to the dilution, it is not absolutely in direct proportion to it.

POISEUILLE'S EQUATION FOR LAMINAR FLOW



$$P_1 - P_2 = \frac{32 U V L}{D^2}$$

$P_1 - P_2$ = Pressure Drop

U = Fluid Viscosity

V = Average Fluid Velocity

L = Length of Pipe

D = Pipe Diameter

Fig. 5

The viscosity of a fluid passing through a tubular system profoundly affects the pressure necessary to force the fluid through the tubular structure. For rigid tubes this is according to a definite physical law, known as Poiseuille's Law for laminar flow. Although one cannot compare the vascular system to rigid tubes, nevertheless to some extent the law may be applicable to the vascular system. Fig. 5 illustrates Poiseuille's Law for laminar flow. Since Poiseuille's Law does not apply for blood in small tubes of less than 0.2 mm in diameter, it cannot be applied to the arterioles. Bingham and Netting have both modified Poiseuille's Law and these more probably apply to the vascular system. Since the lowering of the viscosity affects the peripheral resistance and, therefore, the force of the pump or heart, we suggest that this may be a factor in some of our animal experiments in which uncontrollable oozing took place.

Since it is a well-known fact that a bleeding diathesis may develop in certain patients who have received large amounts of transfused blood, we suggest that the lowering of the viscosity of the blood of such patients resulting from the great dilution of the blood by the citrate solution may be a factor in uncontrollable oozing. We are not unaware, however, that other chemical changes also take place.

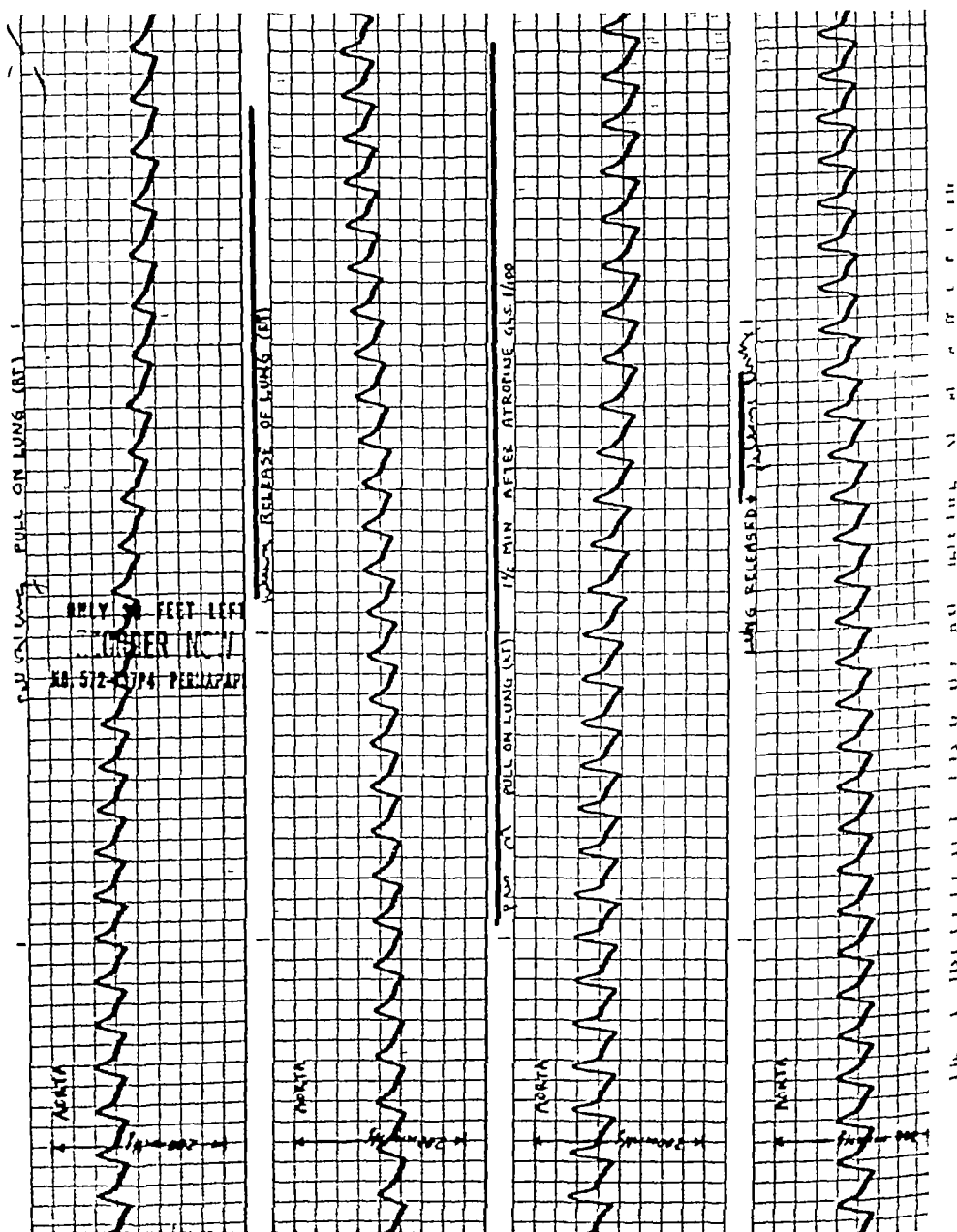
We also wondered if the viscosity of the blood might be changed by the mere mechanical pumping. Viscosity studies were, therefore, carried out on blood which had been drawn from dogs and heparinized. The viscosity was first determined after it was withdrawn and again following thirty minutes of mechanical pumping. We were unable to find any change in the viscosity from the artificial pumping mechanism.

RIGHT-SIDED SUBSTITUTION

Experiments have been carried out in which blood is withdrawn from the right atrium, or vena cava, passes through the pump and is returned to the pulmonary artery thereby by-passing the right ventricle. We picture the right atrial appendage to the vena cava, since with the cannula in the atrium, not only the vena cava blood is withdrawn but also that from the coronary sinus as well. We have been able to successfully substitute for the right ventricle during periods up to 30 minutes. We have determined, during the course of the substitution, both the pulmonary artery pressure and the aorta pressure.

There are profound reflexes present in the dog with regard to the normal atrial distention. In general this is thought to represent the Bainbridge reflex. Briefly stated, the Bainbridge reflex means that a rise in pressure or increased distention of the great veins or right atrium sends up impulses to the brain center causing an acceleration of the heart rate and, therefore, a rise in the blood pressure. Inversely the removal of the normal distention of the atrium or great veins should cause a slowing of the heart and drop in blood pressure. In our experimental animals this reflex, or one similar to it, can be easily produced by mild traction on the right lung. We have noticed this reflex drop in blood pressure constantly with the traction on the right pulmonary artery during its cannulation. Fig 6A shows this profound drop in systemic pressure following traction upon the right lung. The fall in blood pressure begins almost simultaneously with the traction on the lung. Fig 6B also shows a profound drop in systemic pressure as the right sided pump is turned on. It also demonstrates a slowing of the heart rate.

Since it is known that atropine diminishes the nervous impulses taking place by way of the vagi, this drug has been given to determine whether or not this Bainbridge reflex could be abolished. To a large extent, atropine does markedly diminish the lowering of blood pressure due to reflex action. Fig 6C illustrates the fall in blood pressure following traction on the lung and following the administration of atropine. It is seen that the fall is not nearly so great. We believe therefore that the use of atropine to diminish vagal reflexes is a sound one. Such a severe hypotensive reflex is obviously not



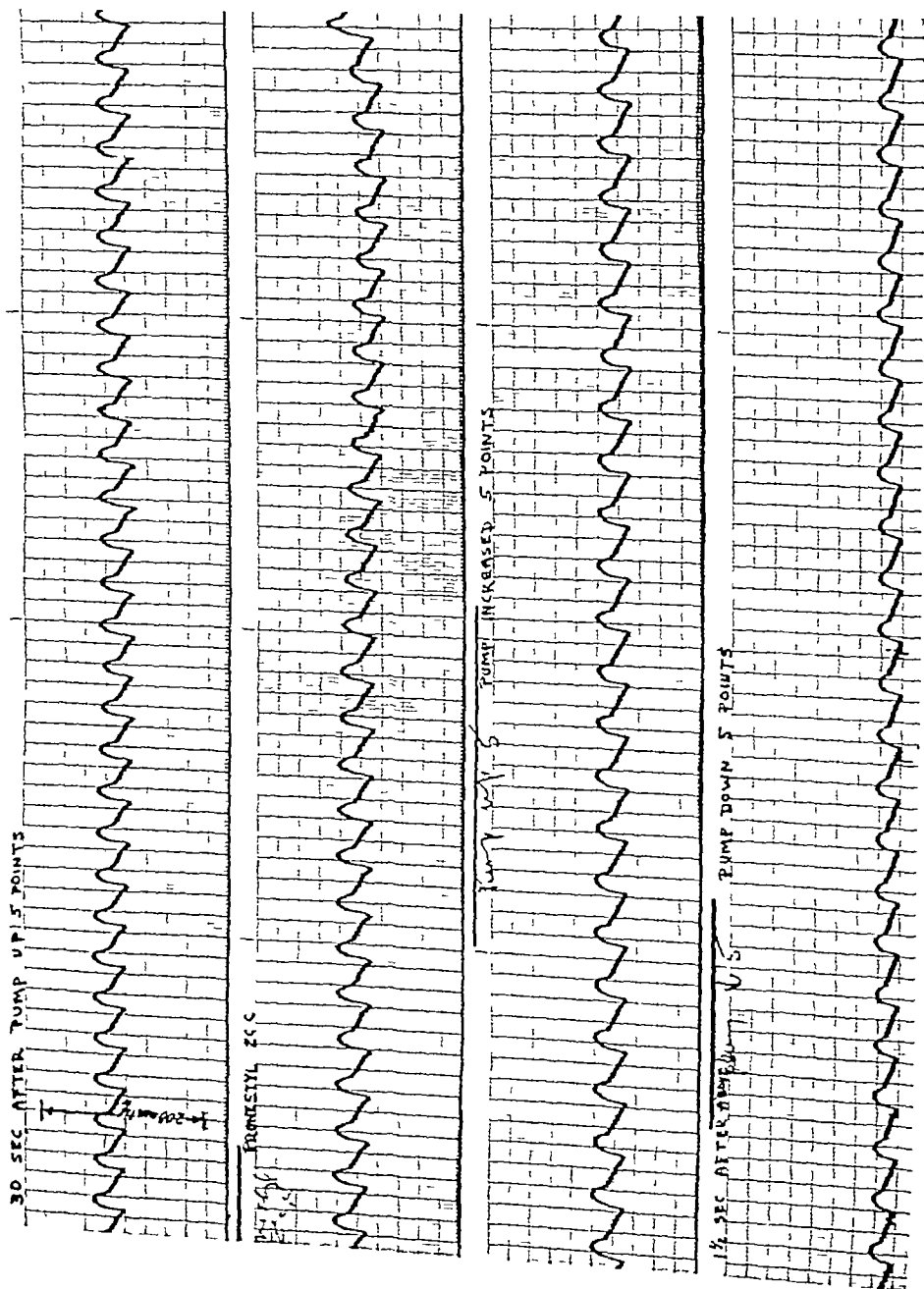
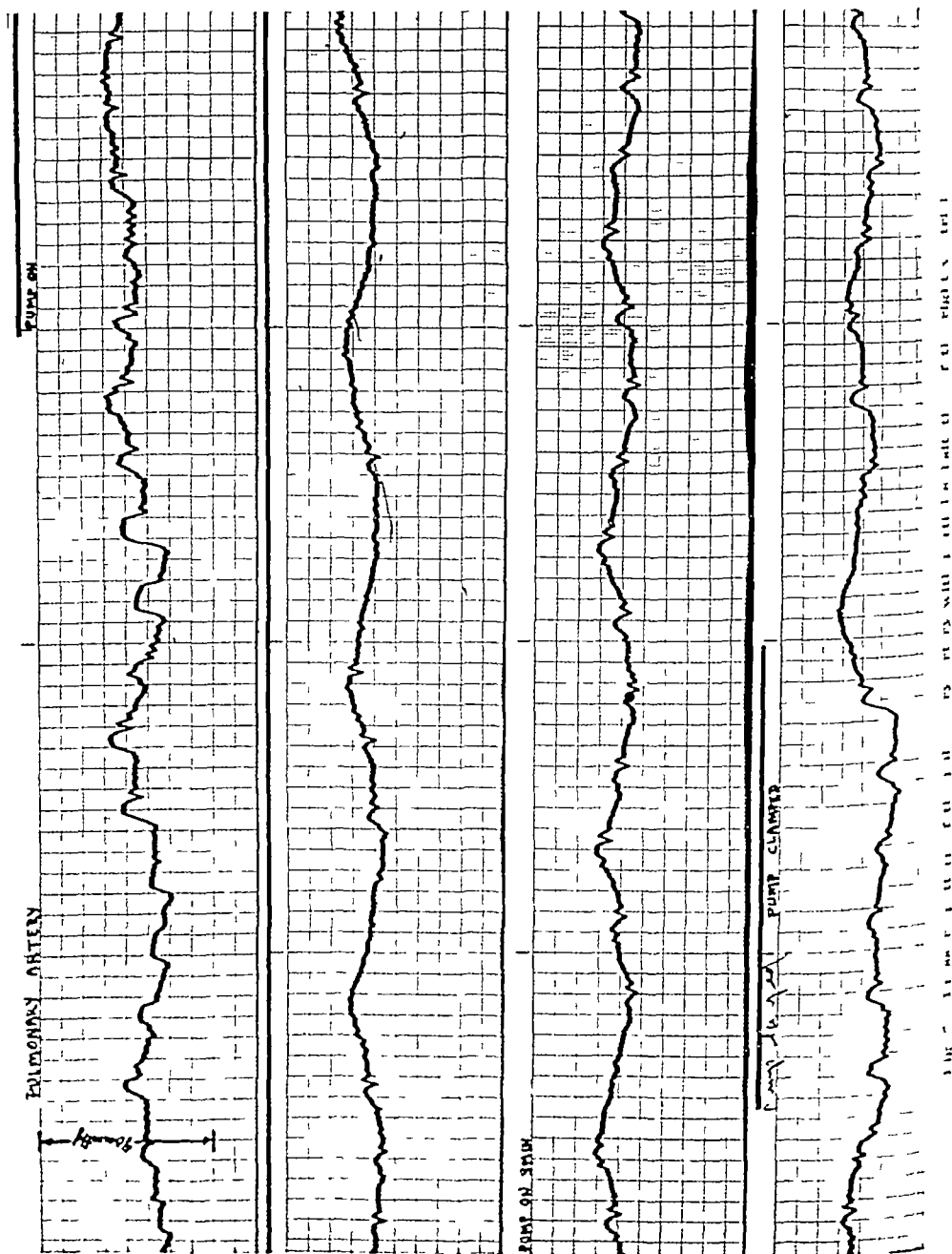


Fig. 6B.—Profound drop in systemic pressure as the right atrium is evacuated also slowing of the heart



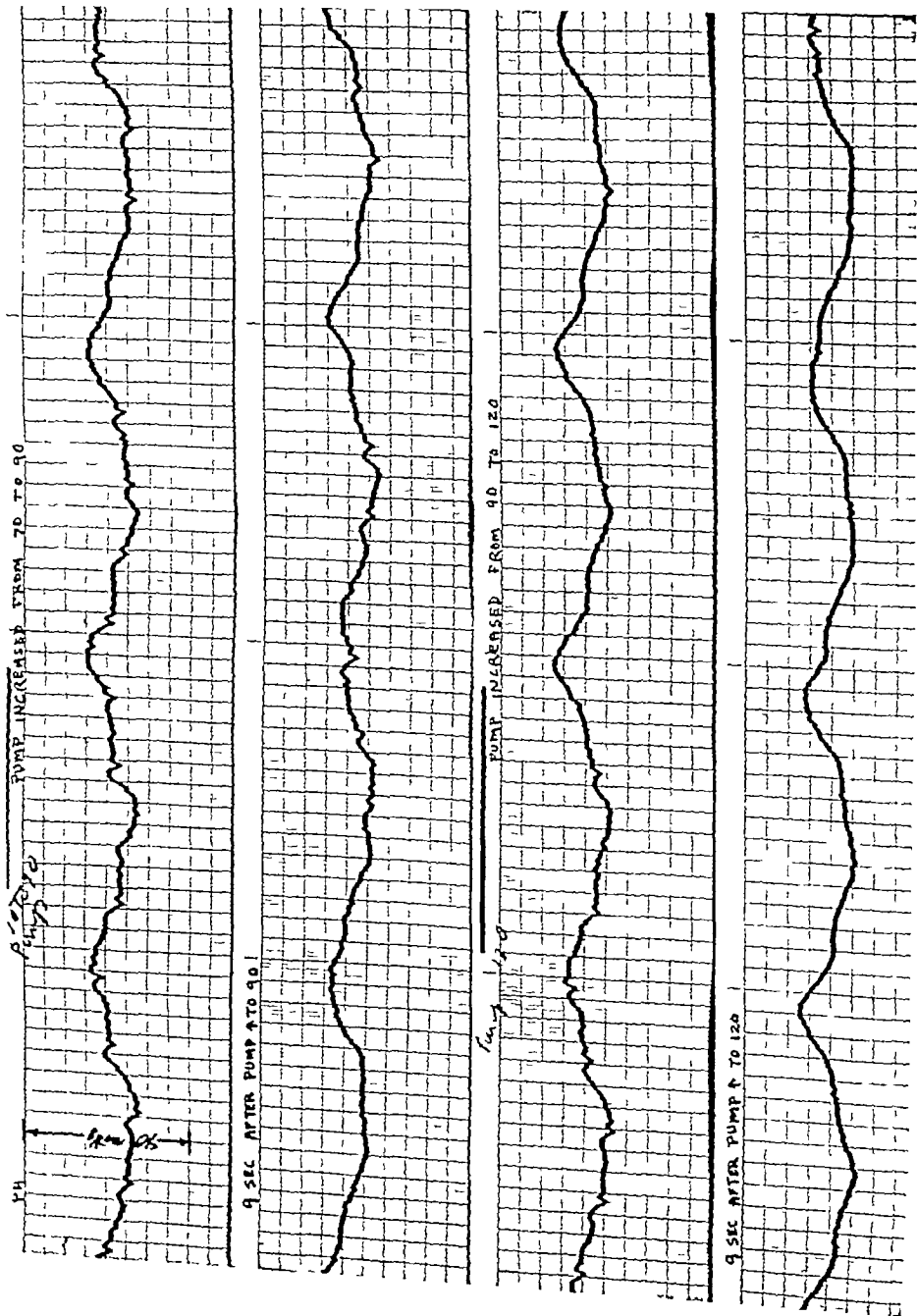


FIG 8 --Pressure pattern of the pulmonary artery showing complete substitution for the right ventricle. The large waves are due to the respirator

present in man as it certainly would be observed during right sided lung resections, especially difficult pneumonectomies where considerable traction is placed on the hilar structures

It has been stated by some investigators particularly Gibbon and associates¹ that the vena cava must be cannulated proximal to the atrium and the cava obstructed between the cannula and the atrium in order to be sure that all the right sided blood is being pumped with the mechanical mechanism. We have found that another method may be used in order to determine if all the right sided blood completely by-passes the ventricle. This is by means of

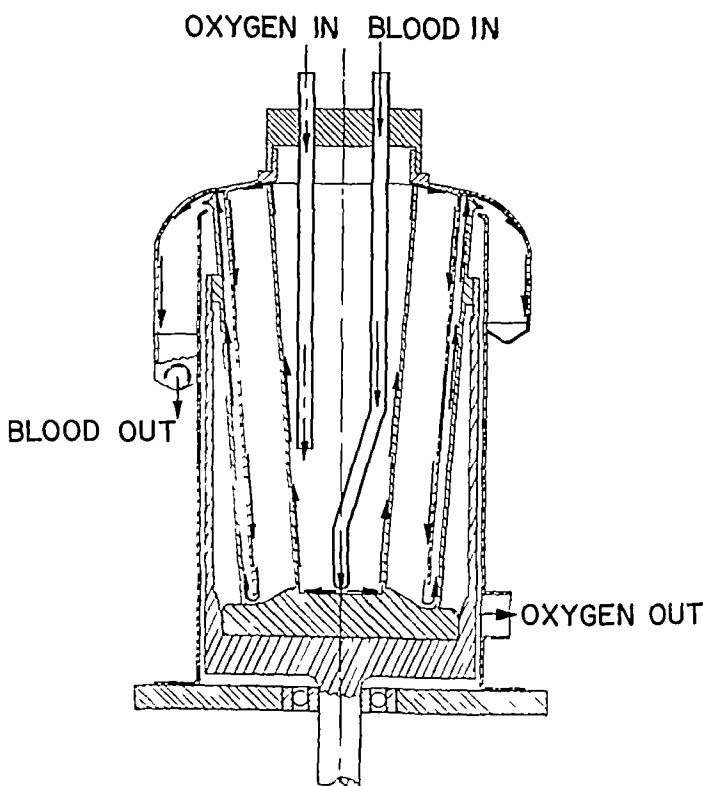


Fig. 9—Rotating cup oxygenator

the pulmonary arterial pressure pattern. As the right sided pump is turned on and blood begins to by pass the right ventricle, there appears in the pressure pattern of the pulmonary artery two types of pressure patterns. One of these represents the ejection of blood from the right ventricle while the other represents the pattern from the mechanical pump during partial substitution for the right ventricle. However, as the mechanical pump pressure is increased and all the blood by passes the right ventricle, the pressure pattern represented by the ventricular beat disappears and the pattern is then solely from the mechanical pump. Figs. 7 and 8 illustrate partial by passing of the right

ventricle and later on complete by-passing of the right ventricle. These findings have been confirmed by either aspiration of the right ventricle or by incising it. Later on in this manuscript we shall describe a similar pressure pattern for the left sided circulation.

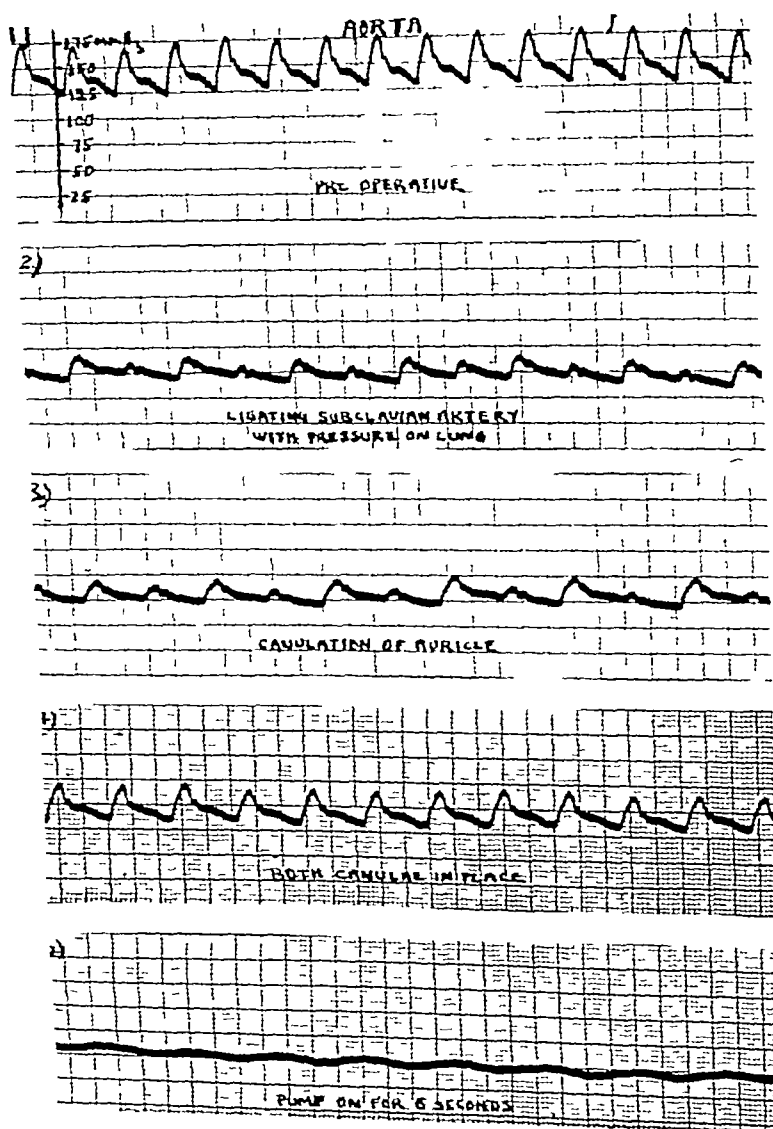


Fig 10—Drop in systemic blood pressure with complete extracorporeal circulation.

COMPLETE EXTRACORPOREAL CIRCULATION

An oxygenator which is illustrated in Fig 9 has been attached to our pump and complete extracorporeal circulation has been carried out. This procedure initiates the same hypotensive reflex as does the right-sided substitution. Fig

completely by-passed. It has been our experience that usually no drop in systemic pressure occurs, although in some experiments we have noted some minor degree of drop in aorta pressure. Atropine will usually abolish the minor hypotensive reflex, if present.

BLOOD CHEMISTRY

During these experiments a large number of tests have been performed on the blood. These have been done before, during, and immediately after the experiments. These include total protein, sodium, potassium, Ph, Hematocrit, blood counts, oxygen saturation, and chlorides. We shall report these changes subsequently.

SUMMARY

1 Sixty-five experiments on dogs using the artificial heart have been performed. These include (a) right sided substitution, (b) complete extracorporeal circulation with mechanical oxygenation, and (c) left sided substitution.

2 No animals have died of infection. This is due, we believe to our type of pump which is sterilized by autoclaving.

3 The viscosity of the blood is profoundly lowered following the administration of large quantities of citrated blood.

4 There are profound atropressor reflexes present on the right side of the heart in dogs which greatly lower the systemic blood pressure, in fact we have found it necessary to support the blood pressure by large transfusions as well as by the administration of vasoconstrictor drugs.

5 This atropressor reflex is not present on the left side in dogs or, at least is not present to the same degree. As a general rule we seldom obtain any drop in systemic blood pressure at all.

6 We present a method of determining when all the blood by-passes the heart. This is by the particular type of arterial pressure pattern obtained.

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DISCUSSION ON "ARTIFICIAL OXYGENATION AND CIRCULATION DURING COMPLETE BY-PASS OF THE HEART," BY DR. JAMES A. HELMSWORTH, DR. LELAND C. CLARK, JR., DR. SAMUEL KAPLAN, DR. ROGER T. SHERMAN AND DR. THOMAS LARGEN, AND "SOME PHYSIOLOGIC ASPECTS OF THE ARTIFICIAL HEART PROBLEM," BY DR. F. D. DODRILL, DR. EDWARD HILL, AND DR. ROBERT GERISCH

DR. JOHN H. GIBBON, JR., Philadelphia—I was delighted to hear these two presentations and to learn that other workers are exploring methods of temporarily diverting the blood stream from the heart. I do not feel competent to discuss the physiologic aspects of the second paper, but I have had some experience with the Clark-Golan lung. In our laboratory we were unable to achieve the good results which were reported by Dr. Helmsworth, but I am sure it was because we did not spend enough time developing and perfecting this method. I think a great deal of work must still be done with this apparatus, e.g., developing automatic control of CO_2 tension and pH, before it can be safely used for total by-pass of the heart in human patients.

I would like to show what has been accomplished with the apparatus we have been using—the new apparatus which was completed last summer. The method of oxygenation consists in the passage of blood over stationary vertical screens enclosed in a plastic case. Oxygen is taken up and carbon dioxide given off by the blood while it is filmed on these screens. Thirteen animals have survived experiments in which the entire blood flow was temporarily diverted from the heart and lungs. These operations were performed between August of 1951 and January of 1952. The entire circulation was carried by the mechanical heart-lung apparatus, both venae cavae being occluded for periods, with the exception of the first one, of more than an hour, the longest for 100 minutes. All of these dogs are alive and perfectly well months afterward. These thirteen experiments were from a consecutive series of nineteen attempts, two of the nineteen animals died from technical errors and the others died several days following the operations.

(Slide) This is a graph illustrating the kind of controls we have. In this dog the entire circulation was carried by the heart-lung apparatus for ninety-six minutes with the blood pressure maintained in the preoperative range. The blood flow was constantly 1,200 mm. per minute, and the pH was well maintained on the alkaline side by the automatic control of the CO_2 tension in the gas exposed to the blood. The arterial oxygen saturation was well maintained and the venous oxygen saturation also remained at a normal level. The latter we regard as indicative of adequate tissue oxygenation and an adequate blood flow. I think control of the pH of the arterial blood is important as Dr. Helmsworth indicated.

When the heart is opened the disposal of the coronary blood flow is relatively easy to accomplish. The coronary venous return is aspirated from the right atrium and continuously returned to the extracorporeal circuit. When I left Philadelphia we had opened the right atrium in four consecutive dogs, for periods between twenty-five and thirty-five minutes. While the right atrium was open the cardiorespiratory functions were maintained by the extracorporeal blood circuit. These four dogs are all alive and well weeks later.

In conclusion I would like to congratulate Dr. Helmsworth upon the excellent detailed report of the results of his experiments. I believe that we are approaching the time when extracorporeal blood circuits of the mechanical heart-lung type can be safely employed in the treatment of human patients.

DR. SANFORD E. LEEDS, San Francisco—I would like to make just two points, one concerning drainage of the right side of the heart. I would like to illustrate the cannula we have been using for this purpose.

(Slide) This was published a couple of years ago (*Proc. Soc. Exper. Biol. & Med.* 75:463, 1950), but I thought it would be well to bring it up again because drainage of the right side was mentioned. This is a 26-gauge right-angle metal cannula which is passed

into the azigos vein, into the superior cava, down through the right auricle and into the inferior vena cava. It is ligated in place so that blood from both vena cavae must pass out through the cannula and none into the right auricle. We have found this quite satisfactory.

The second point concerns other applications of the mechanical heart. (Slide.) We have recently applied it to occlusion of the thoracic aorta. The carotid artery is cannulated and a plastic tube is passed through the femoral artery and into the thoracic aorta. The blood from the common carotid artery is passed into the reservoir cylinder, through the pump and back into the femoral artery via the plastic tubing. By this means the aorta has been occluded for periods up to three hours, with survival. By applying clamps above and below any place in the thoracic or abdominal aorta, the aorta may be resected and anastomosed. The spinal cord receives an excellent blood supply and we have had no cases of instances of spinal cord damage with this type of surgery. We have encountered a good deal of difficulty with heparin and deheparinization. Some of the anastomoses have bled and the animals have not survived.

DR JAMES A HELMSWORTH, Cincinnati—(Closing) I want to thank Dr Gibbon and Dr Leeds for their contributions to this discussion. To bridge the very large gap between total by-pass of the heart in experimental animals and clinical use of the heart-lung machine, many additional studies are required. One simple step to be taken in this evolution of systems to oxygenate and pump the blood is the experimental use of the apparatus for partial by-pass of the heart and lungs in selected patients with disease of the cardiorespiratory system.

We would like to summarize the successful use of the dispersion oxygenator and pump, in a 45-year-old man with extensive fibrotic disease of the lungs and cor pulmonale. His dyspnea, cyanosis, and orthopnea were severe, but the procedure was completed with local anesthesia and without large doses of sedatives. The venous blood was withdrawn through the catheters passed into the inferior vena cava via the saphenous veins, after the blood had been oxygenated and freed of some of its carbon dioxide, it was pumped back into two veins of the right arm. The period of extracorporeal circulation was 75 minutes.

(Slide) The flow through the extracorporeal circuit was slowly increased until it reached 1250 cc per minute. This was equal to approximately one-fourth to one-third of his cardiac output. The blood pressure, which had fallen after the administration of heparin, returned to the normal range. There was an immediate decrease in the rate of respirations and a concomitant relief of dyspnea and orthopnea. The reduction in cyanosis was unequivocal.

(Slide) The control sample of arterial blood for oxygen saturation was satisfactory. In the pumping period the range of saturation was 55 to 60 per cent. After the period of dispersion oxygenation, the arterial oxygen saturation fell into the range of 40 per cent. The control level for this patient's state of cardiorespiratory dysfunction. The plasma carbon dioxide tension fell and the carbon dioxide content and pH rose. There was an increase in the leukocyte count. The erythrocyte count was unchanged and the concentration of hemoglobin remained exactly the same. The quantity of hemoglobin in the supernatant plasma was too small to be determined accurately by routine methods.

The patient experienced definite relief of symptoms during the 75 minutes of partial by-pass. He was given antibiotics for several days and left the hospital without obvious change in the course of pulmonary fibrosis and cor pulmonale.

DR DODKILL—(Closing) I should like to show a 2-minute movie illustrating the exposure of the mitral valve. This was accomplished because we were able to record this particular type of pressure pattern in the aorta and to know that the left ventricle was completely by-passed.

(Movie) This is a by pass of the left ventricle. A suction cannula is in the left atrium, having been placed into it by way of the superior pulmonary vein, and the out flow from the pump is into the aorta by way of the left subclavian artery. A clamp has been placed at the base of the left atrium which is between the suction cannula and the mitral valve. In a moment you will see the mitral valve and, through it, into the ventricle which you see does not contain blood. If we open the base of the atrium when there are still ventricular beats in the pressure pattern, there will be not only a great hemorrhage but also air will be sucked into the pump and death of the animal will quickly occur. We can see some motion of the mitral valve but this is in direct relation to the heartbeat itself and we have not been able to demonstrate independent motion of the valve itself.

TECHNIQUE OF PRODUCING MITRAL STENOSIS OF CONTROLLED DEGREE

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A PRACTICAL technique of creating mitral stenosis for experimental study has long been considered necessary for better understanding of the disease as it occurs clinically. In 1902 Sir Lauder Brunton¹ suggested the feasibility of surgical correction of this crippling disease, emphasizing that such a procedure depended upon mastering of certain technical details. The development of these skillful surgical techniques could only be accomplished in the experimental laboratory. During the following years, many important contributions were made by Beinheim,² Allen,³ Cutler,⁴ Wilson,⁵ Powers,⁶ Katz,⁷ Shaw,⁸ and Connolly⁹ in their attempts to produce mitral stenosis experimentally. Although the results were in general unsatisfactory, significant observations were recorded. Diverse methods to produce a narrowing of the mitral orifice were devised and used with varying degrees of success. These included placement of sutures across the mitral orifice either above or below the coronary vessels, transplantation of pericardium or fascia across the valve orifice, and traumatization to the valve leaflets with simultaneous inoculation with virulent cultures of streptococcus viridans. Connolly recently reported encouraging results. He placed sutures below the coronary vessels in such a manner that they encircled the chordae tendinae so that during systole the valve leaflets closed without regurgitation but during diastole the suture was sufficiently tight to produce a stenosis. Many of these animals died within a few days from acute pulmonary edema but he thought that a significant number survived for a reasonable period of study.

Now that the stenosed valve can be successfully attacked surgically (Harkin¹⁰ and Bailey¹¹), it is of even greater importance that the disturbed dynamics in the disease be better understood. The mechanism of pulmonary edema, once thought to be simply on the basis of back pressure, needs further clarification. Analysis of the factors which produce an increase in the pulmonary bed resistance is greatly needed. These problems can be studied only in the experimental animal with chronic mitral stenosis and back pressure existing over a period of many months or years. The present study is an attempt to produce such an experimental animal.

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Aid is acknowledged by grants from The Life Insurance Medical Research Fund and The National Heart Institute.

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METHODS

The early attempts involved the passage of a small ureteral catheter around the base of the mitral valve. This was accomplished by inserting the catheter beneath the epicardium just above the coronary vessels, gently advancing it until it encircled the valve. A heavy silk suture, tied to the end of the catheter, was then threaded around the base of the valve as the catheter was withdrawn. Later a flexible probe which could be manipulated more easily was substituted for the catheter. A smooth stenosis at the base of the mitral valve (Fig 1) was thus accomplished, but serious hemorrhage occurred in approximately 50 per cent of the cases. In order to minimize bleeding and simplify surgery, a long, straight, round needle threaded with silk or nylon, was inserted through the heart from the posterior aspect, passing just to the right of the interatrial groove and just above the interventricular groove. Each end of the suture was anchored

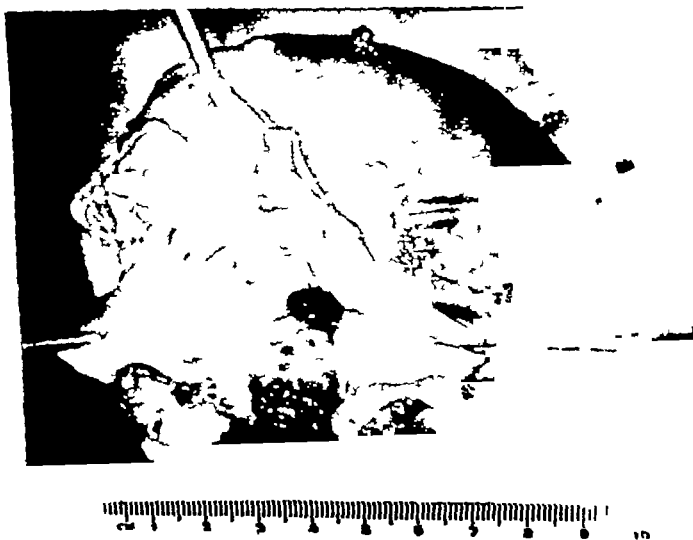


Fig 1—Mitral stenosis produced by advancing ureteral catheter around base of valve. pulmonary edema and death on third postoperative day.

anteriorly in the epicardium just above the coronary vessels. Ligatures so placed were tied tightly enough to distend slightly the left auricular appendage. It was impossible to determine the proper degree of stenosis, however, and the dogs either died within a few hours to a few days from pulmonary edema or remained clinically well indefinitely. Autopsy of the dogs revealed a smooth stenosis at the base of the valve with little or no distortion of the leaflets.

The technique was then modified in several respects and the following method is now used. The animals are premedicated with morphine, using the dosage of 10 mg per kilogram of body weight, and anesthetized with Nembutal, 20 mg per kilogram. An endotracheal catheter is placed and positive pressure respiration carried out by means of a respirator. The left pleural cavity is entered through the fifth intercostal space. The pericardium is opened widely

just anterior to the phrenic nerve and held open by means of traction sutures. Another traction suture of fine silk is placed in the apex of the heart. The index finger of the left hand is placed between the pulmonary artery and the left auricular appendage and then advanced posteriorly until it indents the medial wall of the right atrium. As the heart is displaced out of its pericardial bed, the left thumb indents the lateral wall of the right atrium so that the index finger and thumb approximate the atrial walls. A No. 20 gauge needle, into the end of which a steel wire has been previously anchored and threaded into nylon tubing, is then inserted through the walls of the right atrium passing just to the right of the interatrial groove and just above the A-V groove and

Experimental Mitral Stenosis

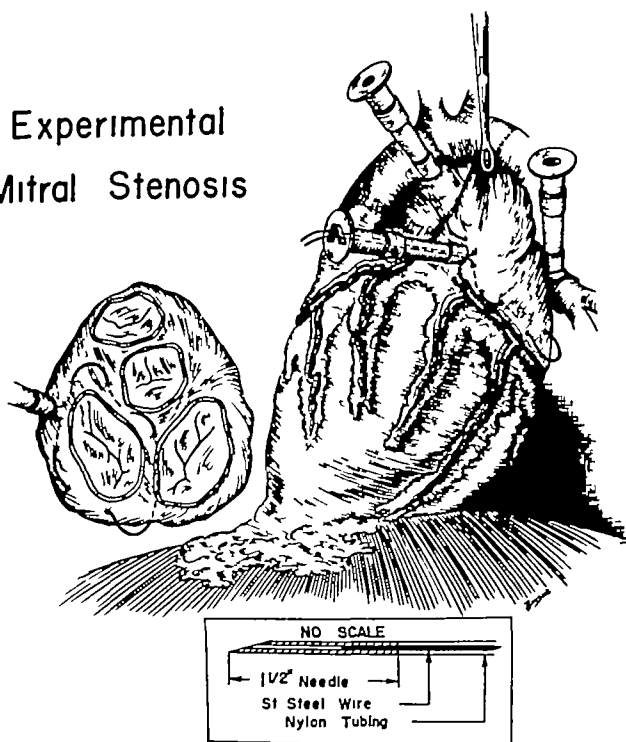


Fig. 2—Drawing illustrating relationship of suture to base of auricular appendage and coronary vessels. Inset at left indicates passage of suture through right atrium. Lower inset illustrates needle.

emerging anteriorly at a similar position. Each end of this suture has been previously prepared with a needle and is anchored in the epicardium just above the coronary vessels. The suture thus occupies a position at the base of the mitral valve (Fig. 2). Controlled narrowing of the mitral orifice was produced as follows. Instead of tying the suture as had been done in the past, it is passed through a cannula whose large, flat head is affixed to the chest wall subcutaneously and whose cardiac end prevents displacement of the heart as the ligature is tightened at a later date (Fig. 3, A and B). The animals tolerate

this operative procedure well. No difficulties were encountered from the standpoint of fibrillation and seldom was there significant bleeding. Occasionally bleeding occurred at the site of passage of the suture into the wall of the right



A



B

Fig. 3—4. Suture has been placed loosely around base of valve and threaded into cannula.
B the size of the mitral orifice is reduced by tightening the suture.

atrium posterolaterally and in these cases hemostasis was easily accomplished by a figure of eight suture of Deknatel No 5-0. In the earlier cases, angiotomy cannulae, which have previously been described (Hamilton¹²), were placed upon the pulmonary artery and the pulmonary vein so that the pressures could be recorded simply by the insertion of long needles into the vessels through the cannulae. Because of the high incidence of infection associated with this large quantity of foreign material, the use of the angiotomy cannulae has recently been abandoned and pressures obtained by means of cardiac catheterization. After the animals have fully recovered from the operative procedure, usually fourteen to twenty-one days, they are premedicated and anesthetized in the same manner. A 1 cm incision is made directly over the head of the cannula, the wire suture exposed and tightened, the suture being maintained in that position by means of a silver clip.

In some of the earlier cases the pulmonary capillary pressure was used as a guide to determine the proper degree of tightening. It was discovered, however, that if the mitral orifice was narrowed sufficiently to produce a significant elevation in capillary pressure, the dogs would not tolerate this degree of narrowing and would proceed rapidly into pulmonary edema and death. Consequently, more recently the degree of narrowing has been determined by palpation of the femoral arterial pulse and the wires tightened until there is a perceptible change in volume or rate. The suture is usually shortened in the range of 1 cm. the first time. A smooth stenosis is thus produced at the base of the mitral valve causing no interference with closure of the valve leaflets. The operative mortality is minimal and the suture has been accurately placed in 95 per cent of the cases.

RESULTS

By means of this technique chronic mitral stenosis has been produced in ten dogs that have survived two and one half to seven months. In one case the suture was improperly placed and partially occluded the tricuspid orifice. All but one of the remaining nine animals developed chronic heart failure. Some were autopsied at varying lengths of time, some were allowed to go downhill and to die from heart failure after a period of observation of several months and others were maintained in a state of chronic heart failure by loosening or tightening the suture when necessary. By this means the stenosis could be controlled and the animals maintained in a state of chronic failure indefinitely. No attempt, however, has been made at this time to maintain a dog longer than seven months. It was found that if the mitral orifice is narrowed suddenly to within the range of 1 cm. in diameter, the dogs develop pulmonary edema and usually die within a few days. On the other hand, if the orifice is narrowed gradually over a period of several weeks, the dogs easily tolerate a stenosis of 1 cm. in diameter without any difficulty. It is estimated that a mitral orifice of 5 to 8 mm. in diameter, produced by gradually narrowing the orifice over a period of several months by shortening the wire suture on two or preferably three occasions will maintain a dog in a state of chronic failure

These dogs develop ascites and edema of the lower extremities at varying periods after tightening. In some cases the onset of failure develops within a week and, if it seems to be progressing rapidly, then the suture is loosened slightly. It has been necessary for numerous paracenteses to be done, five or

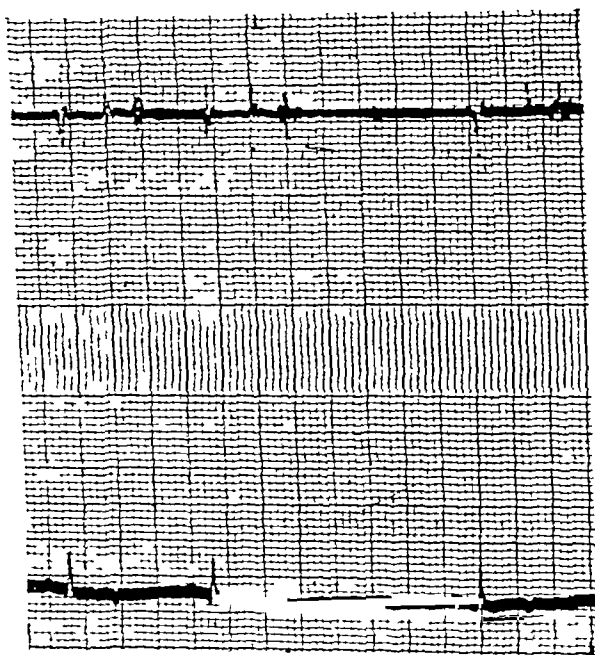


Fig 4—Phonocardiogram of Dog No. 75 showing murmur in early diastole six weeks after tightening of suture.

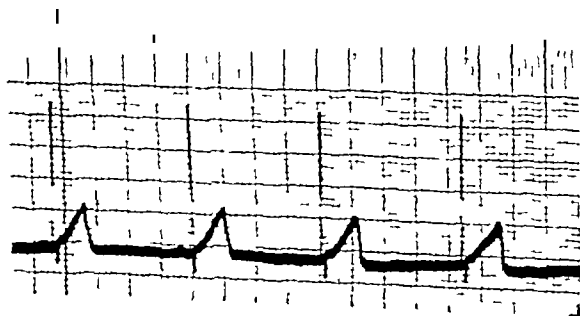
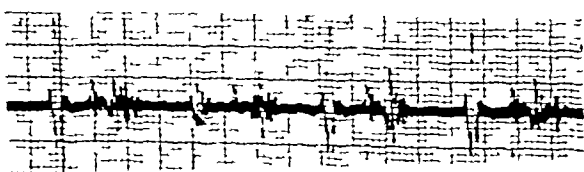


Fig 5—Phonocardiogram of Dog No. 76 showing diastolic murmur two weeks after tightening

six liters of fluid being removed frequently. The dogs have tolerated this state of chronic failure remarkably well. The marked ascites seemed out of proportion to the limitation of activity. In one case ascites developed one week after the first tightening and cleared spontaneously, only to recur several weeks later and persist.

A.



B

Fig 6—Photographs of Dog No. 59. The abdomen is markedly distended with fluid and the lower extremities are edematous five months after tightening of suture.

In all cases a diastolic murmur has been audible. In most cases the murmur could be picked up by phonocardiography (Figs 4 and 5), but occasionally the murmur was so soft that it could not be recorded. It is noted that the murmur is usually in early or mid diastole but never in a presystolic position as is heard clinically.

In regard to the dynamics of the pulmonary circulation, it was observed that in all cases the degree of heart failure seemed out of proportion to the amount of elevation in the capillary and pulmonary artery pressures. Also, none of the dogs developed significant elevation in jugular venous pressure,

thus suggesting the possibility of some mechanism other than back pressure to produce the picture of heart failure. Dog No. 59 survived for a period of seven months in a state of chronic heart failure with massive ascites and edema of the lower extremities (Fig 6, A and B). Pulmonary artery pressures in this case were recorded by means of a needle inserted into a cannula previously placed on the pulmonary artery. During this period of time the highest recorded pulmonary artery pressure was 40/20 mm. Hg, and right ventricular pressure was 40/0 mm. Hg. The day before death this dog had a massive hemoptysis,



Fig 7—Photograph of mitral valve from the auricular aspect (Dog No. 66). Stenosis present for four and one half months. Note smoothness of stenosis and rim of fibrosis on proximal margin.

amounting to approximately 250 cc of fresh blood. On the day of death catheterization revealed a capillary pressure of 15 mm Hg. At autopsy the mitral orifice was found to be 7 mm in diameter and adjacent to the suture a fibrous ring was forming on the endocardial surface. The valve leaflets were uninvolved and when water was injected under pressure into the left ventricle, the valve leaflets closed without evidence of regurgitation. The right ventricular wall was increased to about twice its usual thickness. The dog died in acute pulmonary edema so that the right ventricle and right auricle were markedly distended and all viscera were markedly congested.



Fig 8 A



Fig 8 B

Fig 8—Photographs of Dog No 71 showing distention of abdomen with fluid.

Dog No 66 was killed accidentally at the end of four and one-half months after narrowing of his mitral orifice. No evidence of ascites developed in this dog but he had a pulmonary artery pressure of 30/10 mm Hg with a capillary pressure of 10 mm Hg and right ventricular pressure of 30/0 mm Hg. At autopsy the mitral orifice was approximately 4 mm and it is observed here too that a ring of fibrous tissue developed opposite the suture on the endocardial surface (Fig 7).

Dog No 74 developed evidence of acute congestive failure on the second day after his mitral orifice was narrowed. The suture was loosened and he seemed to recover from his heart failure but three weeks later began to develop ascites and edema. At this time catheterization revealed a capillary pressure of 10 mm Hg, pulmonary artery pressure of 30/12 mm Hg and right ventricular pressure of 30/0 mm Hg. Four liters of ascitic fluid have been removed on three occasions. This dog has a soft, mid diastolic murmur. He has been in a state of chronic failure for three and one-half months and, in spite of the markedly distended abdomen, is active at the present time (Fig 8A and B). Recent catheterization revealed no significant change in pressures.

Dog No 75 developed ascites one month after narrowing of the mitral orifice. Three liters of fluid have been removed twice and two and one-half months after narrowing of this valve he has a pulmonary capillary pressure of 15 mm Hg, pulmonary artery pressure of 40/16 mm Hg and right ventricular pressure of 40/4 mm Hg.

Dog No 76 was tightened on two occasions and three weeks after the second tightening he developed evidence of ascitic fluid. He then went progressively downhill and when it seemed as if he were dying in chronic failure, the wire was loosened slightly. This was followed by marked improvement in his general condition and since then he has been active with a markedly distended abdomen. Catheterization at three and one half months after the initial tightening revealed capillary pressure of 15 mm Hg, a pulmonary artery pressure of 33/15 mm Hg and right ventricular pressures of 30/0 mm Hg.

All cases were studied by electrocardiography. In one case (No 59) there was electrocardiographic evidence of right ventricular strain (ischemic changes) which persisted to the time of death of the animal.

The complications of this technique have been minimal. In one case a thrombus of fairly large size developed around the suture in its course through the right atrium so that the tricuspid orifice was partially occluded.

DISCUSSION

Further study is necessary in order to adequately evaluate problems that have arisen in connection with this technique. Although no evidence of coronary damage has been detected to date, eventually evidence of myocardial ischemia may be noted in the electrocardiogram. These dogs need to be maintained in a state of chronic failure over a much longer period of time in order to adequately evaluate the effect of elevated back pressure on the pulmonary circulation. Additional study is necessary also to further clarify the early development of failure in the presence of minimal elevation of back pressure.

SUMMARY

A technique of producing chronic mitral stenosis of controlled degree in the experimental animal has been described. This technique has satisfactorily produced the picture of chronic mitral stenosis simulating the clinical picture of this disease in man. Further studies are necessary in order to elucidate some of the observations recorded.

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DISCUSSION

DR OTTO C BRANTIGAN, Baltimore—Dr Sim Penton and I have worked on the problem of producing mitral stenosis. After trying many methods without success it was found that rubbing ground glass in No 2 plain catgut suture would produce stenosis if the suture were passed through the valve leaflets. The valves became contracted and scarred with definite excrescences and there was definite narrowing of the valve orifice.

DR ELLISON—(Closing) I would like to thank Dr Brantigan for his comments. Some of the problems that have arisen in connection with this technique still have to be clarified. Whether or not this rim of fibrosis that is developing at the site of the stenosis will interfere with later control of the stenosis we do not know. Since the fibrosis may not develop until four to six months after the valve has been narrowed, it may be that the animals will be compensated by that time and will be in a state of chronic failure, so that further control of the stenosis will be unnecessary.

SURGICAL APPROACH FOR STENOTIC LESIONS OF THE SEMILUNAR VALVES BY EXCISION AND CUSP REPLACEMENT UNDER DIRECT VISION

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STENOTIC lesions of the pulmonary and aortic valves possess certain intrinsic anatomic, pathologic and physiologic similarities.

From the standpoint of construction, both are semilunar valves and both completely depend upon their lateral attachments to the vessel wall for support.

Pathologically, both lesions present the familiar picture of thickening, commissural fusion and fixation of the semilunar cusps with a small central ostium. The valve in most cases of pulmonary valvular stenosis, although firmly fixed, is inclined to be a thin, dome-like structure. In older patients, however, a thicker and more rigid valvular structure is sometimes seen.

In rheumatic aortic stenosis, the inflammatory process produces conglutination of the adjacent cusps at the commissures as well as thickening of the cusps themselves. With organization and scarification, the cusps become rigidly fixed and fused. Often, retraction and severe deformity of the cusps result in associated aortic regurgitation.

Thus, the primary physiologic problem in both cases is an obstruction of ventricular ejection. Experimental data¹ and clinical observations indicate that little alteration of the circulatory dynamics occurs until the outflow tract is reduced to approximately one-fourth its normal size.

This is readily explained by an analysis of the factors involved in the expending of the mechanical energy of the heart. By far the greatest single factor in myocardial work is the energy required to eject a definite volume of blood from the ventricular chambers against a peripheral resistance. A second factor is the energy required to impart velocity to the blood stream.

Ordinarily, calculations of myocardial work neglect the velocity factor since it is assumed that it constitutes but a very small part of the total work of the heart. Normally it amounts to only 1 or 2 per cent. However, in the presence of a stenotic lesion, interference with the ability of the ventricle to

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impart velocity to the blood results in an increase in the velocity factor so that it may rise upward to the magnitude of 50 per cent of the total work of the heart -

Until there is marked obstruction of the valvular orifice (approximately 75 per cent), increased velocity flow readily compensates for the stenotic lesion with only a small increase in cardiac work. With more marked obstruction, maintenance of cardiac output by increased velocity flow results in a marked expenditure of myocardial energy, a situation which eventually results in cardiac failure.

Fig 1, adapted from Evans,² graphically illustrates the effect of semilunar stenotic lesions on myocardial work. The velocity factor, at first negligible, rapidly outstrips the resistance factor.

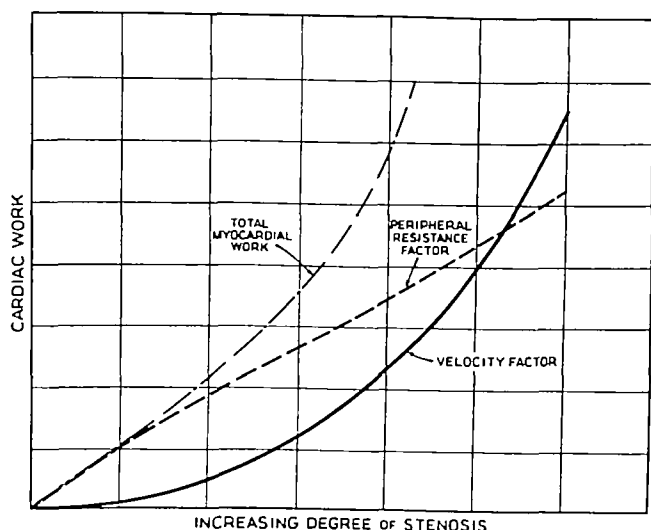


Fig 1—Curves showing the effect of increasing degrees of stenosis on the velocity factor of myocardial work.

Thus, what is required is a return to a higher efficiency area seen below the critical point of obstruction (approximately 75 per cent). With this accomplished, the circulatory dynamics will have returned to a point at which the velocity factor is again small so that the total myocardial work will be markedly diminished.

Because of the basic similarity of pulmonary and aortic valvular stenosis, any consideration of surgical therapy for one lesion would correspondingly suggest a method of approach for the other. This indeed has been the case. The purpose of this communication is to present an approach which is believed to surmount the pathophysiologic problems associated with both lesions.

The technique consists of resection of a pulmonary or aortic cusp and valvular reconstitution by organic cusp prostheses and homograft cusps.

An important factor in these procedures has been the design of instruments which permit resection of one-third of either the pulmonary or the aortic valve (one cusp) under direct vision. Interruption of the circulation is not necessary.

PULMONARY VALVULAR STENOSIS

The first attempt to treat pulmonary valvular stenosis was made by Doven in 1913.³ He attempted to cut the valve by insertion of a tenotomy knife into the right ventricle. The patient survived the procedure but died a few hours postoperatively. Necropsy demonstrated a subvalvular rather than a valvular stenosis.

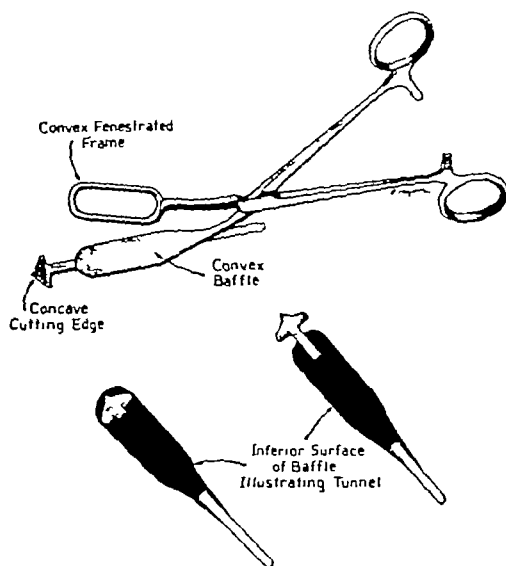
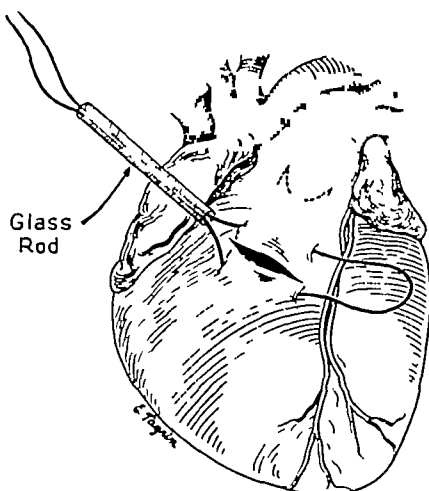


Fig. 2—Instrument used for pulmonary cusp resection and replacement.

Following this, there was a hiatus of over 30 years until the entire field of congenital heart disease was reopened by the monumental report of Blalock and Taussig⁴ in 1945. The ingenious aortic-pulmonary arterial shunt of Potts, Smith, and Gibson⁵ soon followed. Finally, in 1948, Sellors⁶ and Brock⁷ both reported successful direct attacks on the stenotic pulmonary valve by valvulotomy. Recently, experimental⁸ and clinical attempts⁹ have been made to carry out pulmonary valvulotomy under direct vision by means of temporary occlusion of the circulation.

It is clear that surgery done under direct vision is far superior to that done blindly. Equally desirable is an uninterrupted circulation. It has been possible to attack the pulmonary valve with an instrument which accomplishes both of these requisites (Fig. 2). It consists essentially of three parts. The first is a convex solid baffle. Situated above this is a convex fenestrated frame which tightly rims the baffle when the clamp is locked. The third component is a con-

cave cutting edge which directly underlies the baffle in a tunnel, as illustrated. Advance of the cutting edge is limited to a distance of one-half inch. When not in use, a spring mechanism (not shown) retains the cutting edge under the baffle.



TECHNIC OF PURSESTRING SUTURE

Fig 3—For description see text.

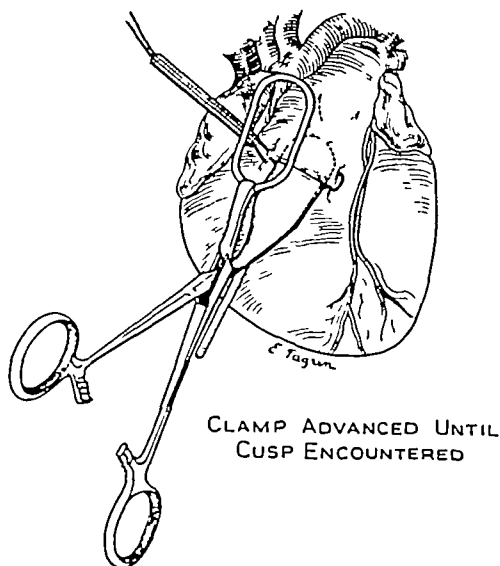


Fig 4—For description see text.

METHOD

Mongrel dogs were used, varying in weight from 7 to 15 kilograms. Intravenous pento barbital sodium was used to obtain anesthesia. Intermittent positive pressure insufflation was administered through an endotracheal tube.

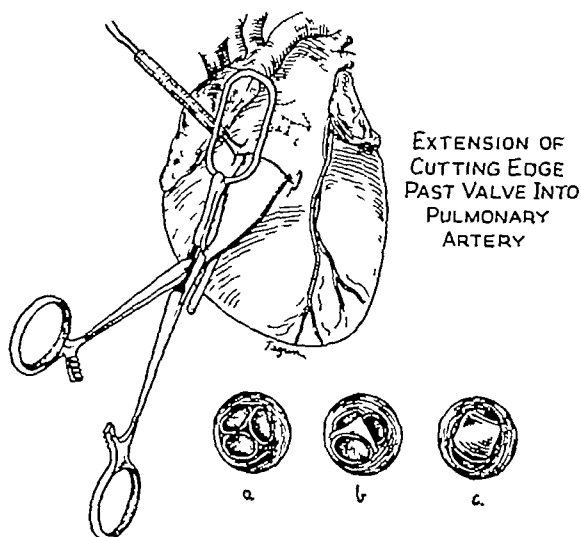


Fig. 5—*a* Pulmonary valve viewed from pulmonary arterial side, *b*, concave cutting edge extended past valve *c* convex baffle advanced past valve

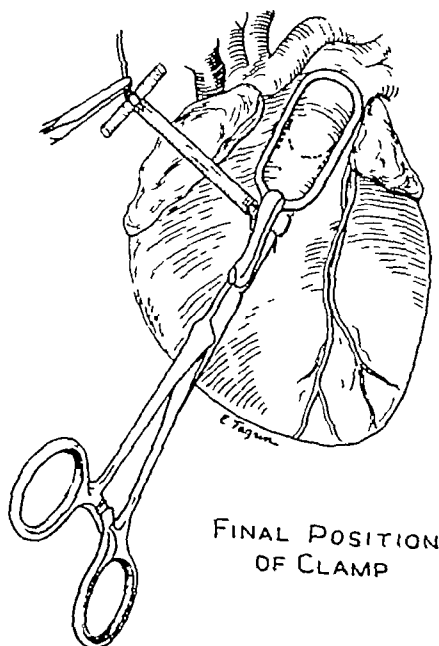


Fig. 6—For description see text.

The chest was entered through the left fourth intercostal space and the pericardium overlying the right ventricle was opened widely. Marsupializing the free edges of the pericardium improved the exposure. A purse string type of suture of 0 braided silk was then placed approximately 1.5 cm from the valve ring (Fig. 3). Both free ends were fed through a small glass tube. We have found that this method permits instantaneous hemostatic control and eliminates the necessity of having two assistants cross stay sutures, with the confusion and blood loss coincident to this maneuver. Sharp pointed Adson scissors were gently pushed through the myocardium between the purse string suture and an incision approximately 1.5 cm long was made. The baffle and its underriding semilunar cutting edge, having previously been moistened with heparin, was quickly introduced into the ventricle and the purse string suture tightened. The baffle was then advanced until the approximate level of the anterior cusp of the pulmonary valve was reached (Fig. 4). At that point, the concave cutting edge was extended past the valve into the pulmonary artery and then retracted.

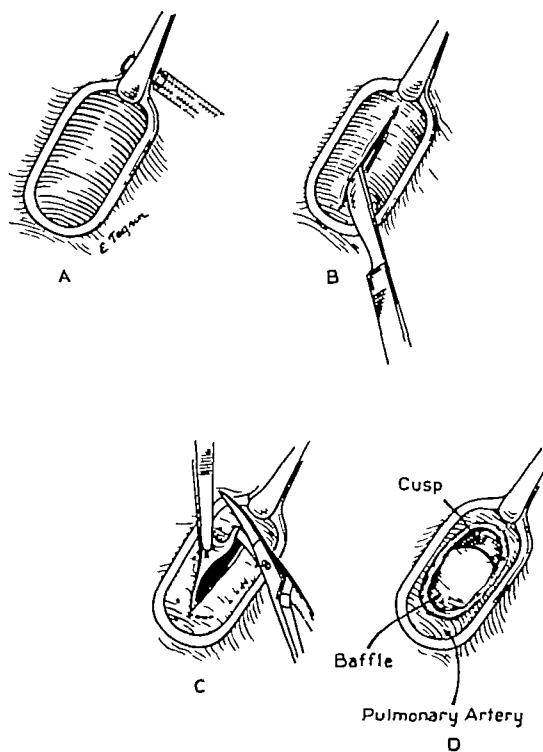


Fig. 7—Technique of excision of pulmonary cusp prior to replacement with homograft cusp. Illustrations drawn from pulmonary arterial aspect.

under the baffle which, in turn, was advanced past the valve. In Fig. 5, *a*, *b*, and *c* illustrate these steps as viewed from the pulmonary arterial aspect. Naturally, in the absence of a valvular stenosis in the dog, the use of the concave cutting edge was quite unnecessary. Its chief value, however, lies in the familiarization of the operator with conditions as they will be seen in the human being. Fig. 6 illustrates the final position of the instrument. The purse string suture was tightened and secured by means of a transverse glass rod and hemostat, as shown. With the clamp locked, the anterior pulmonary cusp and its overlying pulmonary arterial wall were effectively interposed between the underlying baffle and overlying fenestrated frame. Thus, the baffle created an artificial wall in the conus area which permitted excision of

the cusp under direct vision. By virtue of its convex shape, it closely conforms to the luminal curve of the conus so that the outflow tract is unobstructed with the instrument in situ.

The precise technique of cusp excision varied with the two basic methods of cusp replacement employed.

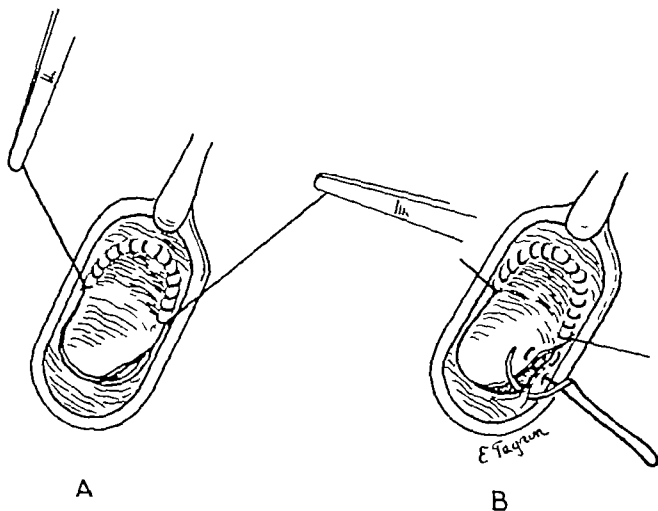


Fig 8—Technique of suturing homograft in place. Two stay sutures have been placed at the superior commissural points of the donor and lateral cusps to insure accurate positioning.

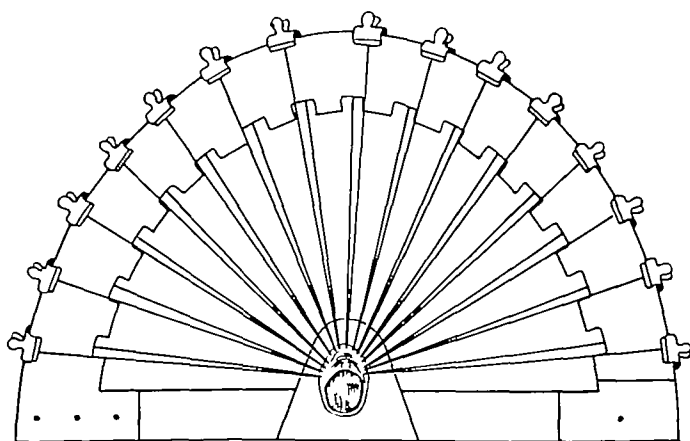


Fig 9—Trimmed aortic cusp homograft. A Annulus B cusp C aortic wall

The first method of pulmonary cusp replacement was the use of pulmonary or aortic cusp homografts. These were obtained from donor animals and consisted of either a pulmonary arterial or aortic segment and an attached cusp.

Fig. 7 illustrates the technique of cusp excision when a homograft cusp was to be inserted. A vertical incision was made over the baffle and the cusp visualized. With the cusp satisfactorily in view, an elliptical segment of pulmonary arterial and right ventricular wall was excised and, finally, the cusp. The homograft cusp was then sutured in place (Fig. 8).

An important point in the accurate positioning of the homograft cusp was the placing of two stay sutures on each side of the donor cusp at its most superior point. By visualizing the superior commissural points of the lateral cusps of the recipient and placing sutures at those points, the graft was placed in exactly the proper position. It is imperative that the superior or lunular edges of the cusps meet exactly. If they do not accurately coapt, regurgitation will be the inevitable result. Failure to carry out this technical point initially resulted in faulty placement and subsequent diastolic murmurs in a number of our animals.



SUTURE PLATE

Fig 10—For description see text.



Fig. 11—Trimmed pulmonary cusp homograft (retouched). A Lip of myocardium below base of cusp B cusp (supported by black paper) C, pulmonary arterial wall

When aortic cusp homografts (Fig 9) were used to replace the resected pulmonary cusp, a method was adopted which involved the least risk of damage to the base of the cusp since it lies in close proximity to the rim of annulus where the suturing must be done. Fig 10 illustrates the trimmed graft (cusp side up) pinned to a piece of cork which is mounted on a fan-shaped metal suture plate. Individual sutures of 00000 arterial silk were taken in the annulus and secured to the suture plate with pinch clips. The graft was then sutured in place with the interrupted technique being used where there was danger of damaging the cusp. A running everting mattress suture of arterial silk completed the suture line (Fig 8). The clamp was then removed and the ventricular incision closed with several interrupted silk sutures.

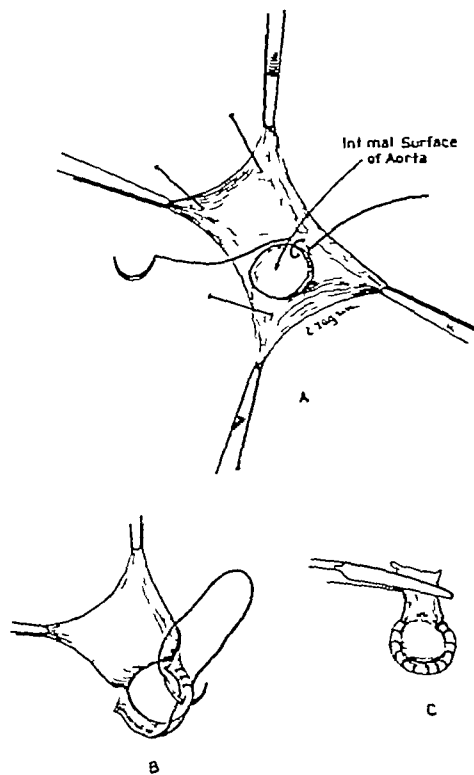


Fig. 12—Method of preparation of the organic cusp prosthesis. A, Aortic ellipse lying on pericardium. B, pericardium sutured to intimal margin of aortic graft. C, completed prosthesis.

A simpler technique was possible with the use of pulmonary valvular homografts. The donor cusp was prepared so that a lip of myocardium was left below the base of the cusp (Fig 11). Therefore, it was possible to rapidly suture the graft in with a continuous type of suture even in those areas close to the cusp.

The second method of pulmonary valvular restoration is the creation of an organic cusp prosthesis fashioned from autograft pericardium and a homograft segment of aortic wall. Fig 12 illustrates the basic steps in the preparation of the prosthesis. The aorta was trimmed to form an ellipse having slightly more length than one half of the external diameter of the base of the pulmonary artery and slightly more width than a single sinus of Valsalva. Utilizing these two measurements, both of which were readily obtained, made the aortic ellipse slightly oversized in both length and width in respect to any single cusp of the pul-

monary valve The adventitia of the aorta was now stripped away A portion of pericardium, free from fat, was then resected and pinned to a board with the serous side down The aortic ellipse was placed on the pericardium with the intimal surface of the aorta exposed A running suture of 00000 arterial silk was employed to suture the pericardium to the margins of the intimal surface of the aortic graft and the knot buried between pericardium and aorta. The excess pericardium was trimmed as necessary The completed organic cusp prosthesis possessed a serous pericardial surface (autograft) and an intimal (homograft) surface

Fig 13 illustrates the various steps in excision of the anterior pulmonary cusp and replacement by the organic cusp prosthesis A transverse incision was made approximately 3 mm distal to the annulus This placed the incision slightly distal to the lunular edge of the

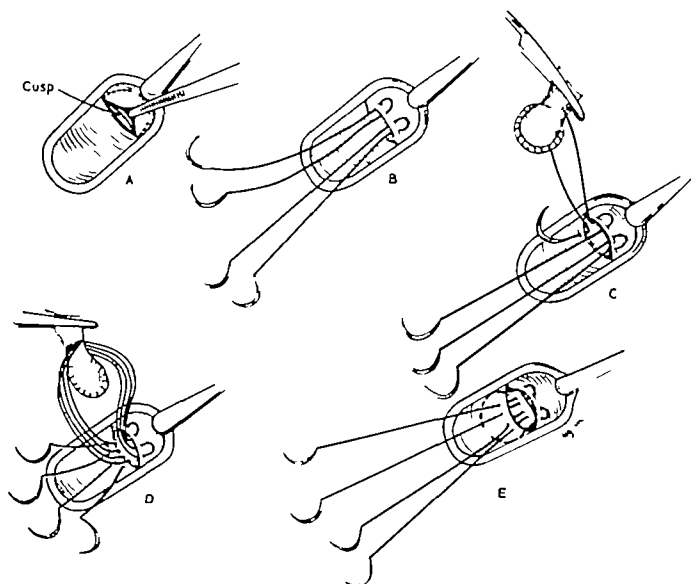


Fig 13—Excision of anterior pulmonary cusp and insertion of organic cusp prosthesis (viewed from pulmonary arterial aspect)

- A Transverse incision distal to annulus revealing underlying baffle and anterior pulmonary cusp
- B Cusp excised and proximal half of horizontal mattress sutures in place
- C and D Sutures fed through pericardium and distal lip of incision
- E Organic prosthesis interposed between pulmonary arterial wall and baffle.

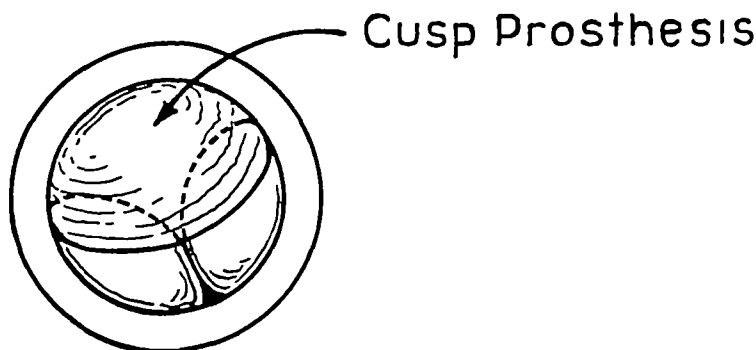


Fig 14—Cusp prosthesis drawn from pulmonary arterial aspect, illustrating method of insuring competency by bolstering effect of unresected cusps

cusps which was then readily visualized and excised. Several horizontal mattress sutures of arterial silk with atraumatic needles swaged on both ends were begun on the proximal lip of the incision (Fig 13B). These were then fed through the pericardium of the graft approximately 4 mm from the edge of the aortic ellipse and then through the distal lip of the incision. By grasping the distal ends of the sutures, the prosthesis was easily pulled into place and slipped between the pulmonary arterial wall and the convex baffle (Fig 13E). The mattress sutures were then tied. Additional interrupted sutures were placed as required and the clamp removed.

Several points bear stressing. The transverse incision must be made slightly distal to the lunular edge of the cusp. This insures free motion of the prosthesis during ventricular systole and competency during diastole, since the oversized prosthesis is supported immediately from behind by the lunular edges of the two remaining cusps (Fig 14).

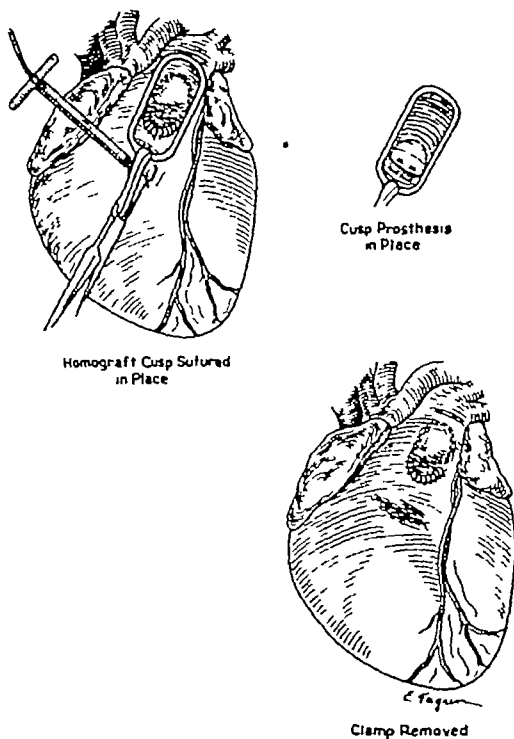


Fig. 15—Illustration showing homograft cusp and cusp prosthesis at completion of operation

A second point of importance is the necessity of leaving a margin of approximately 4 mm. between the edge of the aortic ellipse and the point of entrance of the sutures into the pericardium. An inadequate hinging motion of the prosthesis will occur if the sutures are passed too close to the ellipse. Thus, there will be some obstruction to systolic ejection. On the other hand, too great a distance will permit twisting of the prosthesis on the pericardial pedicle with resultant regurgitation.

Fig. 15 illustrates the homograft cusp and cusp prosthesis at the completion of the operation.

AORTIC STENOSIS

The first attempt to correct aortic stenosis surgically was made in 1912 by Tuffier.¹¹ It was his desire to divide the aortic valve with a knife by entering

the aorta just above the valve ring, but he thought that sufficient experimental data to warrant clinical application were lacking. Therefore an attempt to dilate the stenosed valve was made by invagination of the aortic wall into the ring of the aortic valve. The patient survived and was reported to be still living in 1920.

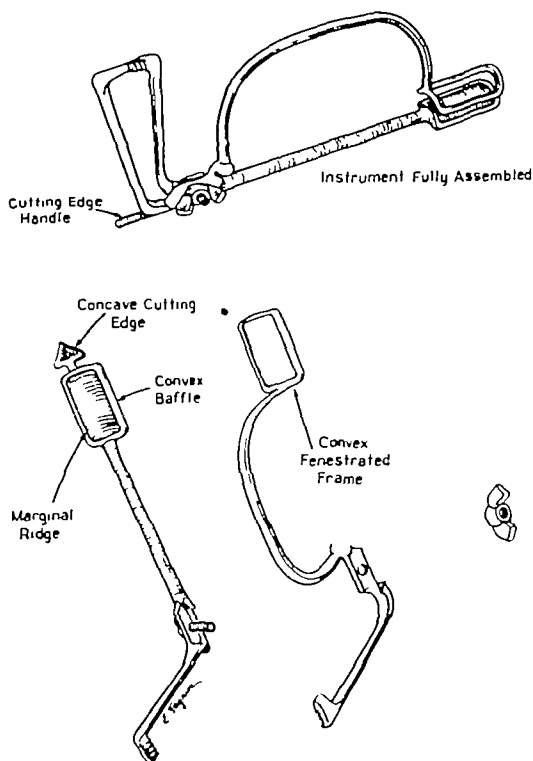


Fig. 16—Instrument used in aortic valve resection and replacement.

The late Dr. Horace G. Smithy carried out extensive experimentation with transaortic and transventricular aortic valvulotomy.^{11, 12}

Recently, Bailey and associates¹³ have described both a transaortic and transventricular method of valvular dilatation and transventricular valvulotomy.

We have attacked the aortic valve utilizing the same approach of cusp excision and replacement as was carried out on the pulmonary valve.

Fig. 16 illustrates the various components of the instrument utilized in aortic cusp resections. The basic principle is unchanged from that seen previously in the pulmonary instrument. As before, there are three basic elements. The first is a convex solid baffle which is rimmed with an elevated marginal ridge. The importance of this ridge will be brought out later. Directly underlying the baffle is a concave cutting edge which is tunneled through the arm proximal to the baffle. Extension of the cutting edge is limited to one-half inch and it is

retained under the baffle by a simple spring mechanism when not in use. The third part of the clamp is a convex fenestrated frame which firmly approximates the marginal ridge of the baffle when the clamp is locked. Its proximal connecting arm is dome shaped. The baffle and fenestrated frame can readily be assembled by the use of a wing nut and bolt, as illustrated.

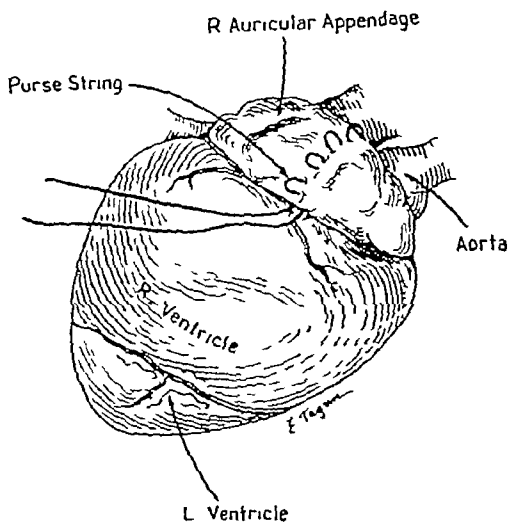


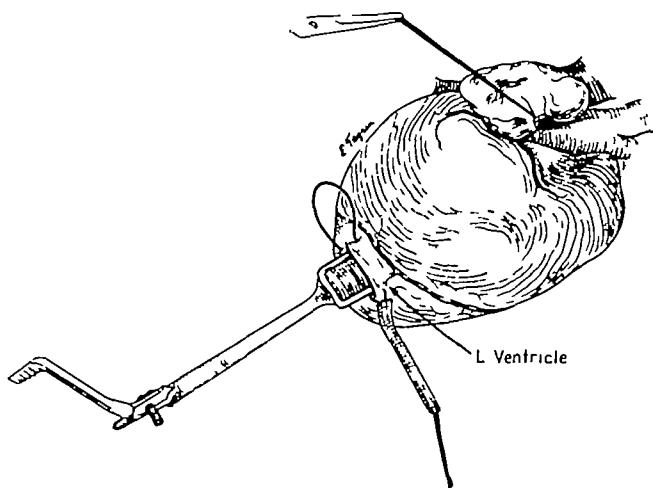
Fig 17—Purse string suture applied at base of right auricular appendage.

METHOD

The chest was entered through the right fifth intercostal space. The sixth rib was divided anteriorly at its costochondral articulation. The pericardium was then opened widely revealing the apex of the left ventricle, the lateral aspect of the right ventricle and the ascending aorta, a large part of which is hidden behind the right auricular appendage. The free edges of the pericardium are marsupialized to improve exposure. A purse-string suture of 00 braided silk was placed at the base of the right auricular appendage (Fig 17). Because of the proximity of the sinoatrial node to the purse string suture, transient arrhythmias were often noted at this point but these were inconsequential. The appendage was then amputated, immediately bringing into view practically the entire ascending aorta. A purse-string suture of 0 braided silk was then placed in the apex of the left ventricle and the ends passed through a glass tube, as described previously. An incision, approximately 1.5 cm long, was then made by sharp pointed iris scissors between the sutures. The convex baffle, the first half of the aortic clamp (with its attached semilunar cutting edge) was rapidly inserted into the left ventricle (Fig 18). The purse-string suture was then tightened and secured in the same manner as was done with the insertion of the pulmonary instrument. The second part of the clamp (the fenestrated frame) was then placed over the area of the posterior (non-coronary) cusp of the aortic valve which directly underlies the posterior sinus of Valsalva. The clamp was maintained in this position by an assistant. The convex baffle and cutting edge were then carefully guided along the interventricular septum toward the posterior cusp as outlined by the fenestrated frame. As the level of the posterior cusp was approached, the semilunar cutting edge was advanced until the baffle had passed through the valve ring. The cutting edge was then permitted to retract under the baffle. The two parts of the clamp were then slipped together and the wing nut applied. The clamp was then locked, impinging the posterior aortic cusp and its overlying wall between the baffle and the fenestrated frame (Fig 19). The cusp may now be excised under direct vision.

Both aortic cusp homografts and organic cusp prostheses have been utilized in reconstitution of the aortic valve. During early pilot experiments in which cusp prostheses were inserted into the aortic valve, it was found that the aorta was held so tightly between the baffle and fenestrated frame that attempts at slipping the prosthesis between the aorta and the baffle always resulted in the aorta being torn. Therefore, the marginal ridge was added to the instrument. This had the effect of depressing the central portion of the baffle and thus provided a slot into which the prosthesis could readily be inserted. The details of each technique are precisely the same as those described previously in pulmonary cusp replacement.

In insertion of the aortic cusp homografts, it was not possible to align the lunular edges of the cusps. This was due to the fact that correct insertion of a homograft cusp requires not only removal of the cusp itself but excision of several millimeters of tissue proximal to the base of the cusp to offset the marginal bit of tissue which must be left for suturing purposes below the base of the cusp to be grafted. Therefore, it was necessary for the instrument to provide visualization of as much as one half centimeter below the base of the cusp. This provided no problem in insertion of homograft cusps into the pulmonary valve since no vital structures lie proximal to the anterior pulmonary cusp. However, attempts to visualize the area below the base of the posterior aortic cusp resulted in impingement of



INSERTION OF 1ST HALF OF CLAMP
WITH CUTTING EDGE

Fig 18—For description see text.

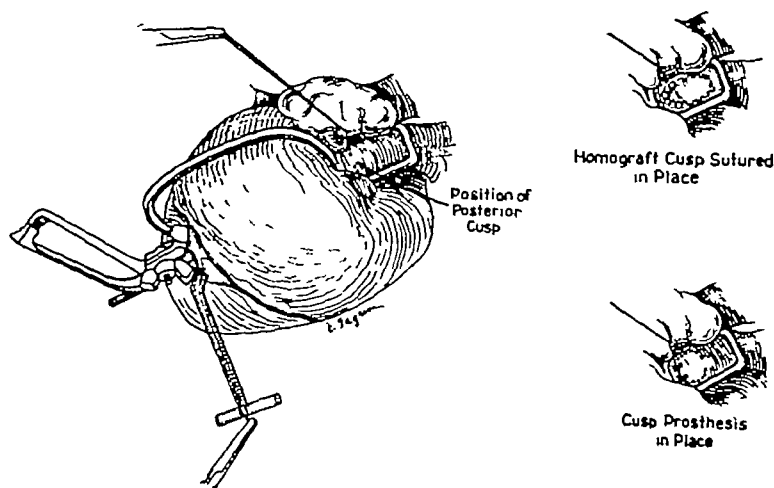
the interventricular septum by the instrument at a point almost directly overlying the atrio-ventricular bundle. Secondly, severe deformity of the right atrium occurred with this maneuver and right ventricular filling was impaired. Almost immediately, a severe bradycardia or bizarre arrhythmia occurred and this was followed by ventricular fibrillation or cardiac arrest.

No such problem was encountered in insertion of organic cusp prostheses into the aortic valve since visualization of the cusp alone is required. Using this technique, no suturing was required at the base of the resected cusp, all work being done at the site of the leading (lunular) edge which could be readily visualized and isolated. The completed procedures are illustrated in Fig 19.

RESULTS OF PULMONARY CUSP REPLACEMENT

Thirty one animals were subjected to pulmonary cusp replacement. The results of these operations are shown in Tables I and II. Twenty two homo-

graft cusps and 9 organic cusp prostheses have been inserted. There were four operative deaths. Three of these were from hemorrhage and one from ventricular fibrillation. There were an additional three deaths due to atelectasis, two from distemper and one from cerebral anoxia due to mechanical failure of the endotracheal cuff.



FINAL POSITION OF CLAMP

Fig. 19—Clamp in position. Note approximate position of posterior cusp. Also shown are the two methods of cusp replacement at operative completion.



Fig. 20—Specimen one and one half months after grafting (Dog 5). There is thickening and fixation of cusp. Positioning of cusp is poor.

The remaining twenty-one animals were sacrificed at intervals varying from five days to ten months postoperatively. In all cases, retrograde perfusion of saline under pressure was used to determine valvular competency. At sacrifice, electrocardiograms and phonocardiograms were taken.

All homograft cusps of animals who died immediately postoperatively and of Dog 3 (sacrificed at five days) appeared delicate and pliable. Valvular competency was observed in each of these animals except Dog 2. In this case, the valve had been placed too low. The homograft cusps which were in place for longer periods of time were totally incapable of functioning as competent cusps, with three exceptions.

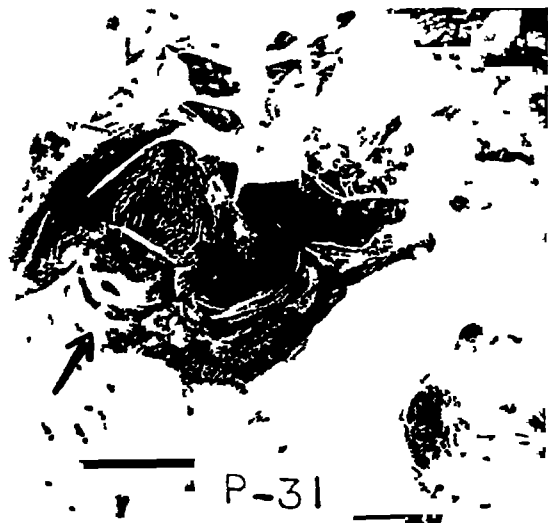


Fig. 21A—Specimen four months after grafting (Dog 31). Note shrinkage and retraction of cusp.

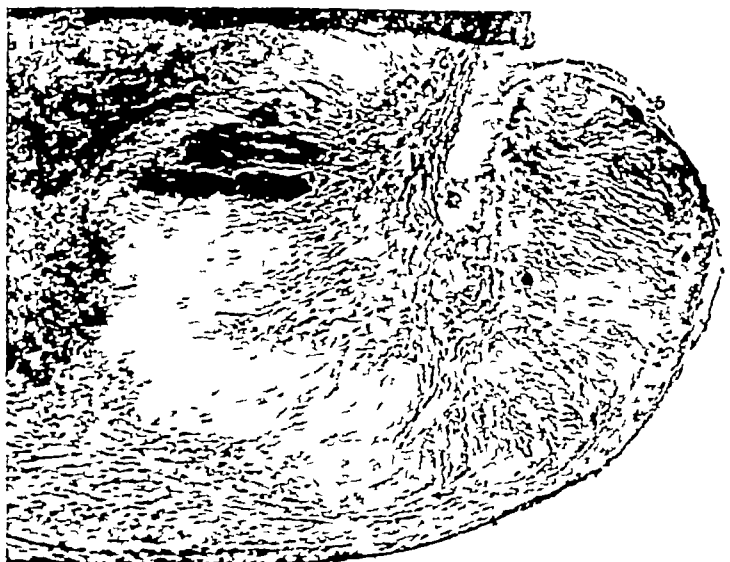


Fig. 21B—Photomicrograph of cusp embedded within connective tissue and undergoing organization ($\times 90$).

The degenerative process appeared to be one of initial thickening and fixation of the cusp due to fibroblastic proliferation (Fig 20). Later, there was marked shrinkage and retraction (Fig 21). The final stage was one in which only the slightest vestige of the cusp remained (Fig 22). The microscopic features of this process are shown in Fig 21B. The cusp was com-

Fig. 22.



Fig 23A

Fig 22.—Specimen ten months after grafting. Only the slightest vestige of a cusp is evident.

Fig 23A.—Specimen seven months after grafting. The cusp is thin and delicate. Directly beneath the graft is an organizing thrombus situated at the sight of the ventricular incision.

pletely embedded within loose connective tissue and organizing blood clot. The leaflet persisted as a necrotic, shadowy outline undergoing organization. There was a marked inflammatory reaction throughout the graft.

In contrast to these failures was the one apparently successful graft of Dog 12 (seven months, Fig 23) The grafted cusp appeared thin and delicate and did not seem to have undergone any gross change Retrograde perfusion of saline demonstrated competency of the valve Fig 23, *B* and *C* shows the microscopic appearance of the grafted cusp of Dog 12 The lines of apposition showed minimal chronic reaction The cusp was delicate, well preserved, and without inflammatory reaction or scarring Special stains showed some slight loss of elastic tissue but the persistence of a fine network of elastic fibrils within the valve leaflet

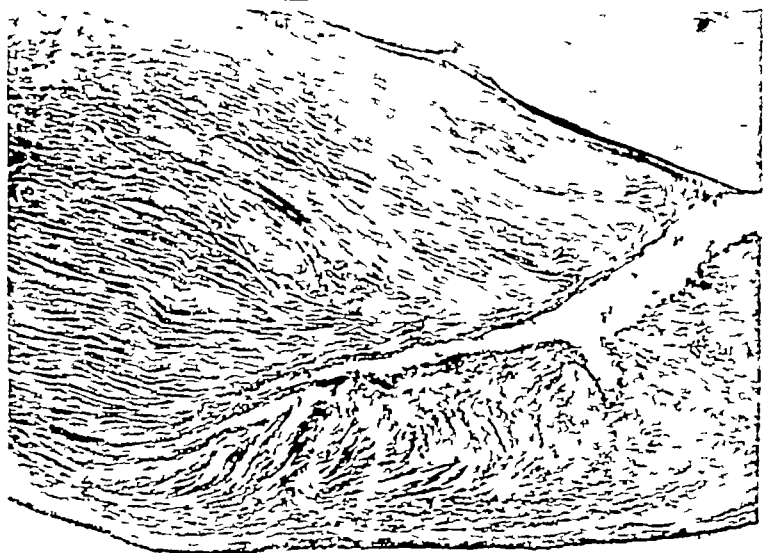
Fig 23*B*Fig 23*C*

Fig 23*B*—Photomicrograph (X40) The cusp is delicate and well preserved
 Fig 23*C*—Higher power photomicrograph (X90) The cusp is without inflammatory reaction or scarring

Animals having pulmonary cusp replacement with organic cusp prostheses were autopsied from three weeks to seven months postoperatively (Table II). All prostheses were well placed and preserved. Only one valve was incompetent. In this animal (Dog 29), the cusp had been made too small and swung freely on its pericardial hinge.

Fig. 24A



Fig. 24B

Fig. 24A—Organic cusp prosthesis of animal sacrificed at four and one-half months. Note excellent cusp.

Fig. 24B—Photomicrograph of prosthesis. Inflammatory connective tissue is present between the aortic graft and pericardium. The aorta is well preserved (elastic tissue stain, x50).

Fig 24 shows a prosthesis four and one-half months after insertion (Dog 30). All the prostheses appeared slightly thicker than they were at the time of surgery. Microscopically, this thickening appeared to be due to the accumulation of inflammatory connective tissue between the pericardium and the aortic graft (Fig 24B). The aortic grafts themselves showed good preservation of structure.

Fig 25A

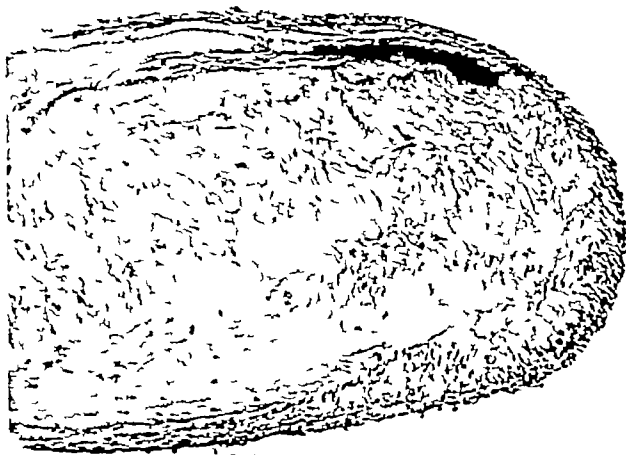


Fig 25B

Fig 25A —Homograft cusp in aortic valve one month after surgery (cusp supported by cardboard for photographic purposes). Note poor positioning of cusp.

Fig 25B —High power photomicrograph. Active connective tissue surrounds and is invading the leaflet.

RESULTS OF AORTIC CUSP REPLACEMENT

Twenty-one animals were subjected to aortic cusp replacement (Tables III and IV). Nine homograft cusps and twelve organic prostheses were inserted.

There were two operative deaths, one from cardiac arrest and one from hemorrhage. An additional two deaths occurred on the first postoperative day due to atelectasis.

The remaining seventeen dogs were sacrificed at intervals varying from one week to four and one-half months. Prior to sacrifice, arterial pressure tracings were obtained using a Sanborn electromanometer as well as electrocardiograms and phonocardiograms. After sacrifice, competency was tested by retrograde saline perfusion under pressure. The autopsy studies corroborated the findings seen in the pulmonary group.

TABLE I. RESULTS OF PULMONARY CUSP REPLACEMENT WITH HOMOGRAFT CUSPS

DOG	CUSP INSERTED	SURVIVAL TIME	PHONOCARDIOGRAM	CONDITION OF CUSP	COMPETENCY AT SACRIFICE
1	Aortic	8.5 mo	Systolic	Shrunken and fibrotic	No
2	Aortic	1 day, atelectasis		Thin and delicate	No
3	Aortic	5 days	No murmur	Thin and delicate	Yes
4	Aortic	10 mo	Systolic, diastolic (?)	Thickened vestige	No
5	Aortic	Operative death, hemorrhage			
6	Aortic	1.5 mo	Systolic and diastolic	Thickened, fixed	No
7	Aortic	1 mo	Systolic	Thickened, shrunken	No
8	Aortic	4 mo, death from dis temper	No examination		
9	Aortic	8 mo	Systolic and diastolic	Thickened vestige	No
10	Pulmonary	4.5 mo	Early systolic	Thin and delicate	Yes
11	Pulmonary	Operative death, hemorrhage			
12	Pulmonary	7 mo	Systolic	Thin and delicate	Yes
13	Pulmonary	1 day, atelectasis		Thin and delicate	Yes
16	Pulmonary	6 mo	Systolic	Thickened vestige	No
17	Pulmonary	Operative death, hemorrhage			
18	Pulmonary	4 mo	Systolic and diastolic	Shrunken, retracted	No
20	Pulmonary	3 weeks	Systolic	Thickened, fixed	No
22	Pulmonary	1 day, cerebral anoxia		Thin and delicate	Yes
25	Pulmonary	3 mo	Systolic	Vestige	No
27	Pulmonary	3 weeks	No murmur	Thin and delicate	Yes
28	Pulmonary	1 day, atelectasis		Thin and delicate	Yes
31	Pulmonary	4 mo	Systolic	Thin and delicate	Yes
				Thin, shrunken	?

TABLE II RESULTS OF PULMONARY CUSP REPLACEMENT WITH CUSP PROSTHESES

DOG	SURVIVAL TIME	PHONOCARDIOGRAM	CONDITION OF CUSP	COMPETENCY AT SACRIFICE
13	7 mo	Systolic	Good	Yes
14	19 days, death from distemper		Good	Yes
19	1 mo	Systolic	Good	Yes
21	3 weeks	Systolic	Good	Yes
23	25 mo	Systolic	Good	Yes
24	6 mo	No murmur	Good	Yes
26	0, ventricular fibrillation as chest closed		Good	Yes
29	2 mo	Systolic and diastolic	Cusp too small	No
30	45 mo	Systolic	Good	Yes

All homograft cusps of the dogs who either died immediately postoperatively or were sacrificed within a short time appeared thin and delicate. Cusps in place for longer periods of time appeared shrunken. All the grafted cusps had been positioned distal to the lunular edges of the recipients' own cusps. Therefore, incompetency resulted from faulty positioning as well as shrinkage of the cusps in the older animals.

Fig. 25 illustrates the typical finding of a poorly positioned posterior cusp in an animal sacrificed one month after operation. Despite the fact that the cusp appeared thin and delicate, the photomicrograph reveals that the cusp is somewhat degenerated with a thin covering of active connective tissue which is invading the leaflet. There is evidence of persistence of necrotic tissue.

Animals having aortic cusp replacement with organic cusp prostheses were sacrificed at intervals between one week and four and one half months (Table IV). All prostheses were well positioned. Competency was observed in all animals except Dog 4. Clinically, the animal appeared listless almost immediately after operation and was sacrificed twenty-one days later. Examination revealed that the aortic wall was fragmented directly overlying the prosthesis and that a perforation had occurred into the right atrium with the creation of an aortatrial fistula. The cusp was overlying the fistula and was covered with a thrombus.

TABLE III RESULTS OF AORTIC CUSP REPLACEMENT WITH HOMOGRAFT AORTIC CUSPS

DOG	SURVIVAL TIME	PHONOCARDIOGRAM	POSITION	CONDITION OF CUSP	COMPETENCY AT SACRIFICE
1	1 mo	Diastolic	Poor	Shrunken	No
2	1 day, atelectasis		Poor	Thin and delicate	No
3	3 mo	Systolic	Poor	Shrunken	No
5	1 day, atelectasis		Poor	Thin and delicate	No
6	1 week	Systolic	Poor	Thin and delicate	No
7	1 mo	Systolic and diastolic	Poor	Shrunken	No
9	2 mo	Systolic and diastolic	Poor	Shrunken	No
18	1 mo	Systolic	Poor	Thin and delicate	No
19	1 mo	Diastolic	Poor	Thin and delicate	No

TABLE IV RESULTS OF AORTIC CUSP REPLACEMENT WITH CUSP PROSTHESES

DOG	SURVIVAL TIME	PHONOCARDIOGRAM	CONDITION OF CUSP	FUNCTION
4	3 weeks	Systolic and diastolic	Poor	No
8	4 1/2 mo	Early systolic	Good	Yes
10	Operative death, cardiac arrest			
11	Operative death, hemorrhage			
12	3 mo	Systolic	Good	Yes
13	1 1/2 mo	No murmur	Good	Yes
14	1 week	Systolic	Good	Yes
15	1 mo	No murmur	Good	Yes
16	3 weeks	Systolic	Slight ballooning	Yes
17	1 mo	Systolic	Good	Yes
20	1 mo.	Systolic	Good	Yes
21	4 1/2 mo	Systolic	Good	Yes

Fig 26A

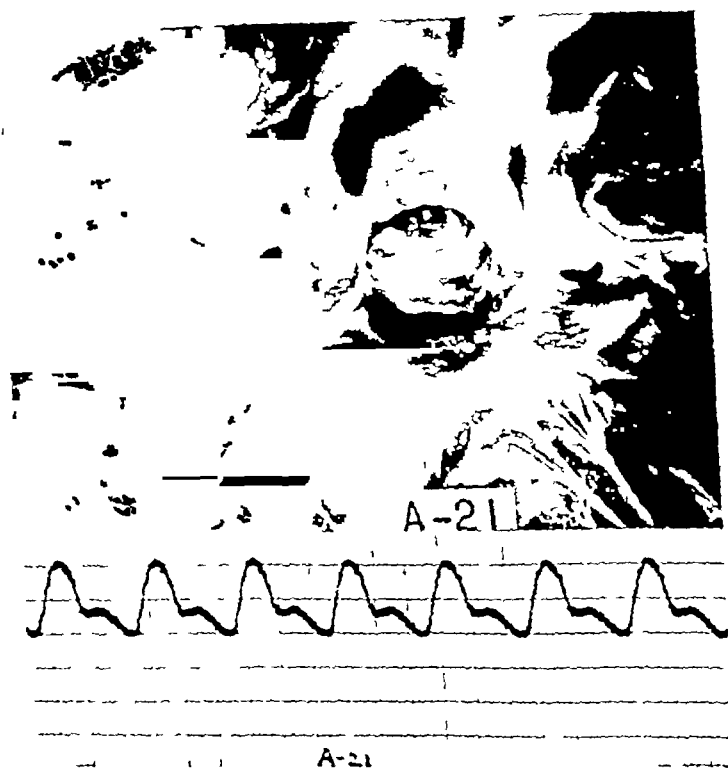


Fig 26B

Fig 26A—Cusp prosthesis in aortic valve in animal sacrificed four and one half months after surgery.

Fig 26B—Aortic pressure tracing, done prior to sacrifice. There is no evidence of insufficiency or systolic obstruction.

In all other animals the prostheses appeared satisfactory in all respects. They all appeared slightly thicker than at the time of insertion and, microscopically, this again was demonstrated to be a result of the presence of inflammatory connective tissue between the aorta and the pericardium.

Fig 26A is a photograph of an organic cusp prosthesis inserted four and one-half months prior to sacrifice. The cusp is slightly thicker than at the time of surgery. The arterial tracing (done at the time of sacrifice) is seen in Fig 26B.

DISCUSSION

To date, intracardiac surgery of pulmonary and aortic valvular stenosis has been confined to blind incision and dilatation of the valve. Functional restoration, therefore, is dependent upon residual flexibility of the valve. If the pathologic process has rendered the valve rigid and calcific, these procedures may produce only limited improvement. Thus, the limiting factor is the state of the valve itself.

Resection of a portion of the valve would permit permanent maintenance of an adequate outflow tract regardless of the condition of the valve. It is axiomatic, however, that any partial valvular resection cannot be carried out at the expense of creation of a regurgitant lesion. Experimental¹ and clinical¹² data have shown that the effect of a suddenly imposed aortic regurgitant lesion is highly detrimental and is disastrous in the diseased human heart. It is probable that a moderate amount of surgically imposed pulmonary regurgitation may be tolerated. That this may not always be the case is illustrated by the report of Glover and associates.¹⁴

It would seem, therefore, that surgical excision of a portion of the valve combined with adequate means of control of regurgitation is a logical approach to the problem of stenotic lesions of the semilunar valves. The advantages of doing the surgery under direct vision with uninterrupted blood flow are apparent.

The results with the use of homograft pulmonary and aortic cusps have been disappointing and are in agreement with the reports of Bill and associates,¹⁵ Hufnagel,¹⁶ and Lam and associates.¹⁷ While the degenerative process may vary in terms of time, the end result is fibrosis and shrinkage of the cusp. It is difficult to speculate upon the reason for the apparently successful grafting of Dog 12 (Fig 23.1).

The use of the combined autograft and homograft cusp prostheses has been more successful in the time observed. All the specimens appeared slightly thicker than they were at the time of insertion but seemed to meet all the requirements for functional cusps. Retrograde perfusion studies and arterial tracings demonstrated that they could adequately prevent regurgitation. Since it is possible to create any desired size of prosthesis, one might speculatively suggest the application of this method for the control of associated regurgitant lesions, especially of the aortic valve, since incompetency so frequently augments the problem of the valvular stenosis.

Many questions remain to be answered. The effectiveness of the operation on the diseased human heart remains to be determined. Moreover, the long term efficiency of the autograft homograft prostheses is still unknown.

SUMMARY

- 1 An approach for the surgical relief of stenotic lesions of the semilunar valves is presented
- 2 Instruments are described which permit excision of one-third of either the aortic or the pulmonary valve (one cusp) and adjacent vascular wall This may be done under direct vision without interruption of the circulation
- 3 Valvular reconstitution has been carried out with homograft aortic and pulmonary cusps and also organic cusp prostheses
- 4 The use of homografted cusps is entirely unsatisfactory
- 5 The organic cusp prostheses functionally act as competent flap valves

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EXPERIMENTAL PULMONARY VENOUS OCCLUSION

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THIS report presents a study of the survival or death of dogs subjected to division of the pulmonary veins from one lung. It has been thought that a fatal result uniformly follows such complete unilateral division of the pulmonary veins. However, in 1940, Hyndman and associates,¹ studying the effect of pulmonary congestion on tuberculosis in the dog, remarked that ligation of all the veins on the left at one operation was "not in the least incompatible with life." In 1948, Swan and Mulligan² reported the effects of division of pulmonary veins in dogs. These investigators observed that healthy dogs survive indefinitely following division of the pulmonary veins from one lobe. However, complete ligation of the pulmonary veins from one lung was fatal in two dogs which they reported. One of these animals died twenty-four hours after division of the left pulmonary veins, the other died three days after division of the right pulmonary veins. These latter authors reasoned that dogs with lobal ligation survived whereas those with total unilateral ligations died because of a quantitative difference between the two procedures related to the volume of blood loss as pulmonary hemorrhage.

Our own unreported experience with lobal vein ligations corroborated the excellent studies of Swan and Mulligan. However, it was noted in other similar experiments that certain dogs survived even when the venous return from an entire lung was divided. Being unaware at that time of the work of Hyndman and associates,¹ it was presumed that penicillin might have exerted a favorable effect on the course of these animals. The present study was performed to provide a quantitative assessment of the hypothesis that penicillin exerted a mitigating effect on the mortality rate in pulmonary venous occlusion.

METHODS

Healthy, adult mongrel dogs were used, varying in weight from 6 to 14 kilograms. They were fed on stock diet of dry dog biscuit and water and kept in individual cages throughout the experiment. Anesthesia was induced with intravenous sodium pentobarbital after preliminary medication with morphine and atropine. The total dosage of sodium pentobarbital did not exceed 30 mg/kg of body weight, most of this was given intravenously until the dog became unconscious, after which the animal was usually given a small additional intramuscular dose. Pulmonary ventilation was maintained by intermittent insufflation of oxygen through a sterilized endotracheal tube with inflated

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off. In a few of the earlier animals in the series small amounts of ether vapor were employed for maintenance. All of the animals awoke promptly at the conclusion of the operative procedure and were allowed diet and water as desired. The rapidity with which normal eating habits were resumed was of some prognostic significance, a dog which was eating normally within one or two days usually recovered.

"Aseptic silk" technique was employed throughout. The chest was entered by an intercostal incision on either side usually through the fifth intercostal space. The pulmonary veins were isolated extrapericardially at the hilum of the lung and were divided between transfexion ligatures. Closure of the chest was performed in layers without drainage. At the conclusion of the procedure a single hypodermocentesis of normal saline solution was given in the amount of 30 cc/kg of body weight. No further supportive therapy was employed except in the animals of the penicillin-treated group. The agent used was crystalline penicillin 'G' in a dosage of 20 000 units/kg of body weight. It was given intramuscularly at the conclusion of operation and at daily intervals thereafter until the animals resumed their diet and were clearly on the way to recovery. Administration of penicillin was continued for a period of two to five days with an average of three days.

In ten animals blood cultures were taken and maintained under both aerobic and anaerobic conditions. Autopsies were performed on all animals promptly after death. Confirmation of the completeness of pulmonary venous interruption was secured in all instances. Photographs of the gross specimens were made and sections were taken from representative areas of the lung for histologic study.

RESULTS

Twenty-five dogs were subjected to complete division of the pulmonary veins from one lung. This procedure was performed on the left side in eight animals and on the right side in seventeen. The animals were divided into four groups, the variables being the side of operation and the employment of penicillin therapy after operation.

Group I, Complete Division of the Left Pulmonary Veins With Postoperative Penicillin—There were four dogs in this initial group. Soon after division of the veins, the lung became dark red and congested but the animal showed no untoward general signs and the immediate postoperative condition was similar to that following simple thoracotomy. The early postoperative days were marked by less than usual spontaneous activity, by a mild cough, and production of small amounts of bloody sputum. Within five days, however, all animals appeared well and penicillin had been discontinued; one dog received the antibiotic for only one day after operation. The dogs were sacrificed at varying periods up to thirteen months after operation.

Comment—The benign clinical course of these animals was at least partially attributed to penicillin. To evaluate this factor a second group of animals was studied.

Group II, Complete Division of the Left Pulmonary Veins Without Penicillin—Four dogs made up this group, similar in all respects to the preceding except that penicillin was omitted. All of these animals survived the division of all pulmonary veins on the left side with essentially the same clinical picture presented by those in the penicillin-treated group. At the time of sacrifice, from 5 days to 6 months after operation, the dogs appeared quite well.

Comment It was obvious that the beneficial effect of penicillin could not be assessed in animals with ligation of the left pulmonary veins, since this procedure was unassociated with any mortality under the conditions of our experiments. In an attempt to impose a more severe strain on the experimental animal, a third group of dogs was studied.

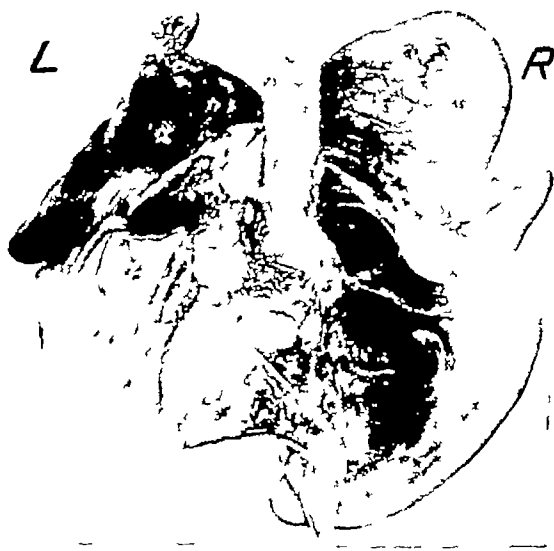


Fig 1—Photograph of dog's lung 20 hours after division of right pulmonary veins. Death occurred despite penicillin therapy. Note severe congestion in left upper lobe as well as in entire right lung.

Group III, Complete Division of the Right Pulmonary Veins Without Penicillin—This group comprised nine dogs, six of which died in from one to ten days, with an average postoperative life of three and a half days. These animals showed a clinical picture of sluggishness, anorexia, and a persistent cough productive of large amounts of bloody, mucoid sputum. In some animals there was extreme orthopnea in the terminal state. In others, increasing torpor was the most prominent feature. One animal appeared on the way to recovery after a week, but developed signs of severe infection and died after ten days with widespread bronchopneumonia in the lung on the operated side. Multiple small abscesses were present in the periphery of this lung. Three of these animals had bacteriologic studies of the blood, all cultures were positive.

Three dogs with all of the right pulmonary veins divided did not die although their postoperative course was quite stormy. In two of these animals

blood cultures were taken, one had a single positive culture on the day after operation and the other had bacteriologic proof of septicaemia on five occasions during the first two weeks following surgery. Both are alive several months after ligation.

Comment This group of animals demonstrates that under the stated conditions there is a high but not invariable mortality associated with total interruption of the right pulmonary veins. It appears likely that infection may play a part in the unfavorable course of these untreated animals. The positive blood cultures also suggest a logical reason for the use of antibiotic therapy.

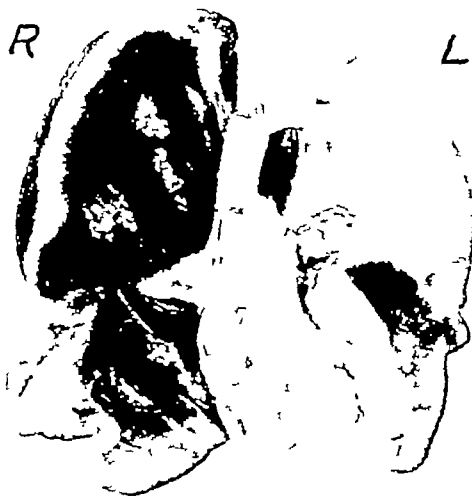


Fig. 2—Thoracic viscera of dog sacrificed 24 hours after division of right pulmonary veins. Intense congestion of right lung is present.

Group IV, Complete Division of the Right Pulmonary Veins With Post-operative Penicillin—There were eight animals in this group of which seven survived. In the one dog which died after 20 hours, the entire right lung was congested as would be expected, but there was in addition a similar involvement of the left upper lobe (Fig 1). It is possible that this additional involvement, presumably due to atelectasis from inhaled secretions of the contralateral lung, led to quantitative insufficiency of ventilated alveoli. A single blood culture in this animal was negative. The picture in this animal may be compared with that seen in an animal sacrificed for pathologic study 24 hours after division of the pulmonary veins from the right lung (Fig 2). In this latter animal, which was progressing satisfactorily, the entire right lung was solidified but there was no comparable change on the left. A portion of the left lower lobe was slightly congested but still air-containing. At 10 days after operation there is no sign of gross alteration in the opposite lung, as seen in Fig 3. The right lung is beginning to clear in its lower portion.

This process of clearing goes on steadily, as seen in the lungs of a dog sacrificed one month after interruption of the right pulmonary veins (Fig 4). The affected lung is now more than half expanded, with intervening areas of atelectasis and incomplete resolution of h  morrhagic congestion. The process in the entire lung varies but little from that which has been known to occur during resolution of congestion in single lobes. Details of the mechanism of clearing will be reported elsewhere.³

By 10 weeks after ligation of the right pulmonary veins in penicillin treated animals, there is fairly good pulmonary expansion (Fig 5). This is complete in the lower portion of the lung but cannot be demonstrated in the upper portion because of tears in the pulmonary parenchyma associated with division of adhesions over the right upper lobe. After 6 months the lung is fully an containing but somewhat contracted with a moderate degree of diffuse interstitial fibrosis (Fig 9).



Fig 3—Lungs of dog 10 days after division of right pulmonary veins. Congestion of right lung beginning to clear at lower portion.

The histologic changes at various periods after ligation are correlated with the gross appearance of the lung. After 24 hours (Fig 6) the pulmonary parenchyma is stuffed with red cells and leukocytes so that the normal architecture of the lung is barely discernible. By 10 days after ligation (Fig 7) the pleura shows an organizing fibrinous exudate, and macrophages filled with hemosiderin are prominent in the unaltered pulmonary parenchyma.

After 1 month (Fig 8) the alveolar ventilation is much improved with focal areas of fibrosis distributed widely throughout the parenchyma. After 6 months (Fig 9) the picture is one of complete aeration but there is diffuse thickening of alveolar septa.

Comment When penicillin is given after total interruption of the right pulmonary veins the mortality rate (12.5 per cent) is appreciably less than in the untreated group (66.6 per cent). These groups are small, and the mortality figures may be taken as indicative of a trend rather than as an absolute index of antibiotic efficacy in pulmonary venous congestion.

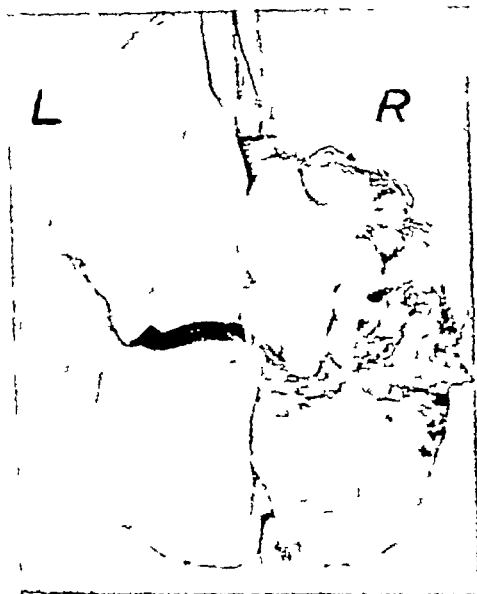


Fig. 4—Lungs of dog 1 month after division of right pulmonary veins. Pale areas of right lung represent aerated portion with darker intervening areas of atelectasis and congestion.



Fig. 5—Ten weeks after division of right pulmonary veins there is expansion of lower portion of right lung with collapse of upper portion due to tear of pulmonary parenchyma associated with division of adhesions during removal. Note thickened pleura over interlobar fissure.

DISCUSSION

It is apparent that ligation of all the pulmonary veins from one lung of the dog, while extremely disturbing in many instances and fatal in some, is nonetheless survived by certain animals. All of our animals have survived when this procedure was performed on the left side. When the right pulmonary veins are totally interrupted, however, the mortality rate has been quite high, possibly because the operative insult is inflicted on an appreciably larger volume of pulmonary tissue. It has been known that ligation of the veins from a single pulmonary lobe is tolerated by the dog. We have corroborated the observation



Fig 6—Division of right pulmonary veins 24 hours previously. Note intense congestion of pulmonary parenchyma with erythrocytes. Alveolar outlines are indistinct (Hematoxylin and eosin $\times 210$)

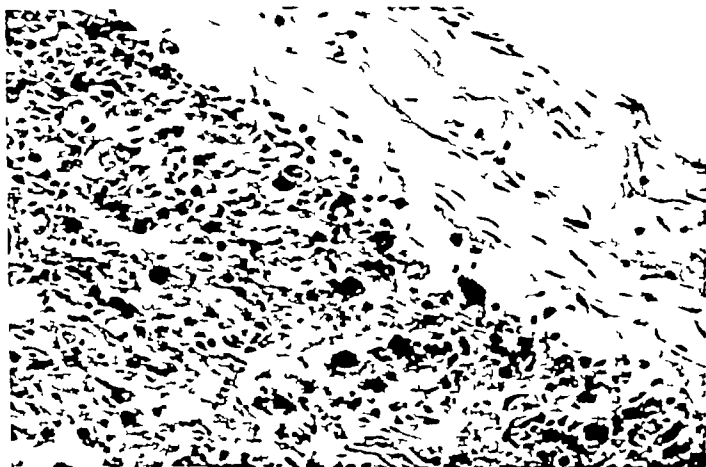


Fig 7—Division of right pulmonary veins 10 days previously. Note fibrinous pleural exudate and hemosiderin filled macrophages in unacrated alveoli (Hematoxylin and eosin $\times 380$)

that the entire left lung may be similarly treated without overtaxing the compensatory mechanisms by which the animal usually adjusts to such venous interruption

If one administers penicillin postoperatively to those animals whose right pulmonary veins have been interrupted, the survival rate is increased, for reasons which are not immediately obvious. It would appear, however, that

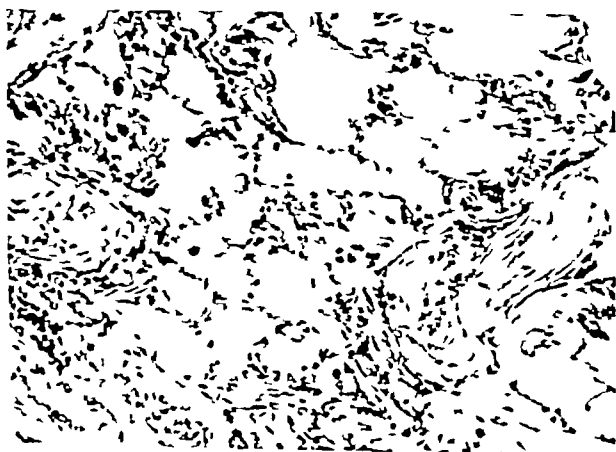


Fig 8—Division of right pulmonary veins 1 month previously. Widely distributed areas of focal fibrosis with intervening well-ventilated alveoli

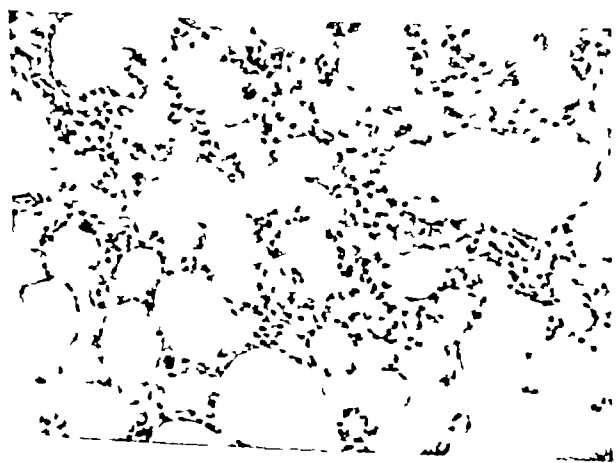


Fig 9—Division of right pulmonary veins 6 months previously. Alveoli completely aerated but there is diffuse interstitial fibrosis.

penicillin had altered in some way the usual mechanism resulting in death of these animals

Three possible explanations of this alteration may be mentioned. First, it is apparent from gross and microscopic examination that a large amount of blood is trapped in the congested lung. Such local fluid loss might well lead to shock, and the increase in weight of the affected lung is consistent with this hy-

pothesis. Although all of our animals received a single hypodermoclysis of normal saline solution after operation, this would not be considered vigorous shock therapy by usual standards. Fluid depletion might well be severe in the ensuing days and critical evaluation of this has not yet been attempted. The moderate depression of the hematocrit in those dogs which survived for a few days does not suggest that acute anemia plays a highly significant role.

A second possible explanation of death in these animals may lie in ventilatory insufficiency. The lung on the side of operation is rendered unfit for respiratory exchange by the blood which fills its parenchyma and overflows into the trachea as bloody sputum. Such sputum may be aspirated into the opposite lung and result in further interference with respiratory exchange until finally the animal dies of anoxia.

Both of these mechanisms—decreased blood volume and respiratory insufficiency—demand critical appraisal, but it is not clear how the beneficial effect of penicillin could be explained if these were the main cause of death in experimental venous occlusion. Penicillin might be expected to have its chief effect on the third possible factor—infection.

That infection plays some role is evidenced by the finding of a positive blood culture in many of the untreated animals and in only one of the treated animals. The origin of these organisms is not clear although there is little doubt that the congested lung provides an excellent medium for their growth. The inhibition of bacterial growth by penicillin might be adduced as the reason for increased survival in treated animals with occlusion of the right pulmonary veins. But if infection is the sole determining factor, it seems strange that it does not exert a similar detrimental effect on the animals with left-sided venous occlusion.

From the data at hand, no positive conclusion may be drawn regarding the precise mechanism of death in our animals. It seems probable that a combination of local fluid loss, ventilatory insufficiency and infection may be responsible.

There are two clinical examples of pulmonary venous interruption to which these studies may be relevant. Ligation of pulmonary veins has been advised in the treatment of pulmonary tuberculosis and has been given limited clinical trial by Edel,⁴ Kerschner,⁵ and O'Shaughnessy.⁶ In the unlikely event that this method of venous congestion should be more widely used, the role of antibiotics in minimizing its untoward effects would be significant. As a more common problem, if the venous drainage from a major pulmonary area is accidentally compromised at surgery, some have advised removal of the affected segment. If the studies reported here may be applied to patients, such advice would seem unduly radical. On the other hand, preliminary physiological studies suggest that a lung which reexpands after such venous congestion is not capable of significant respiratory exchange but is primarily a space occupying structure. Further study may be expected to clarify the functional status of such a lung.

SUMMARY

Interruption of the entire pulmonary venous return from one lung in dogs is compatible in some instances with prolonged survival. Mortality from such a

procedure is decreased when penicillin is given after operation. The mechanism of this reduction in mortality is discussed. The physiologic implications of the problem are under study.

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DISCUSSION

DR F A. HUGHES, Memphis.—We have produced pulmonary venous congestion in the dog experimentally, in relation to pulmonary implantation.

(Slide.) Here is a dog that survived implantation of the left lower lobe. The reason I show this is because this dog has complete occlusion of the veins, pulmonary and all others, and occlusion of the bronchus of the left lower lobe.

(Slide.) The left lower lobe is congested and distended.

(Slide.) Here are the sites of anastomoses of the artery and the vein, and you see the vein is completely occluded, the bronchus was completely occluded also. The artery is not well anastomosed but remains patent. This dog was killed three weeks after operation. The specimens are from the parenchyma of the lobe, this is the normal lobe and this is the lobe subjected to implantation. There is enormous dilatation of the bronchi here.

(Slide.) Here one cannot see the site of anastomosis or an aneurysm around the vein.

It might be possible for patients to survive easily if a vein were obstructed. I should think that that portion of the lung should preferably be removed. Dr Hanlon has shown that, in some instances, it would probably be justifiable to give adequate doses of antibiotics.

DR RALPH D ALLEY, Albany, N Y.—Dr Burke has shown a definite correlation between survival rate and administration of antibiotics. He has not explained the difference in mortality on the two sides, and I would like to suggest that the 100 per cent survival on the left side might be related to the anatomic fact that the venous drainage from the right subcardiac or accessory lobe is usually into the left inferior vein. A ligation about the left inferior vein inadvertently placed distal to the right accessory venous inflow might serve as a venous vent to the nonligated side and explain the high survival rate on the left side.

The principal reason for my arising, however, is to stress the importance of an hemodynamical variable not mentioned in the control of these experiments. It has been known for many years that the bronchial arterial collateral system of the lungs undergoes great expansion in association with chronic pulmonary disease states. This fact was emphasized by Dr Emil Holman in the early 1930's and more recently, Dr Averill Liebow of New Haven has depicted the changes very strikingly with plastic bronchovascular cast. Very little concerning the bronchial veins has been described beyond the fact that they do exist and drain both into the pulmonary veins and into the azygos system. Recently in an as yet unpublished work, Dr Liebow has also shown an expansion of the bronchial venous system accompanying chronic pulmonary disease states. In the light of this knowledge experiments involving the pulmonary circulation are necessarily incompletely controlled unless the bronchial vascular system is taken into account.

For example, although "healthy mongrel dogs" were utilized in these experiments, we do not know their medical background. The usual batch of laboratory animals represents a cross sectional sampling of the full spectrum of canine socioeconomic groups with, if anything, a more generous proportion of former canine vagrants. The latter, under conditions of privation earlier in their careers, may have had episodes of distemper and pneumonitis which left their mark of an expanded bronchial vascular system which, by virtue of the expansion of that portion of the bronchial venous bed flowing into the azygos system, may have had a safety valve to cope with the conditions of Dr Burke's experiments.

DR OSLER ABBOTT, Atlanta—I wonder if I might be permitted to draw attention to some other experiments which have been carried on in this line by Nature, namely, in the form of disease. The two primary ones I think of are, first, the extremely severe and tight mitral stenosis, in some of which, as you know, it is extremely difficult to see at autopsy how the venous return to the lung got to the left ventricle. Second, is the occasional patient with bronchiectasis in which the resected specimen is characterized by a long standing and well recognized thrombosis of the pulmonary vein of the segment or lobe. I think it offers a very important clue, because in those instances there has been observed considerable dilatation and varicose formations of the bronchovenous system. I think another point is that we must stress the susceptibility of such areas of the lung, or any organ with impaired circulation, to infection. This work, then, emphasizes the extreme importance of control of pulmonary suppuration in mitral stenosis. I wonder if I might suggest to Dr Hanlon the advisability of applying Cellophane prepared by Dr Hufnagel's technique to the pulmonary venous system, to stimulate Nature's slow progressive experiments.

DR HANLON—(Closing) I would like to thank the discussants for their remarks. With regard to the comments of Dr Alley, we are cognizant of the anatomy and the anomalies thereof on the left side. Although Dr Burke did not stress it, the anatomic completeness of the occlusion on the left side was confirmed in all the nonsurviving animals at the time of postmortem examination. As regards the socioeconomic factors, I fear I must confess that the social service aspects of our experimental laboratory are rather inadequate at the present time, so that the previous history of these animals is, as he pointed out, unknown to us. With regard to Dr Abbott's suggestion, I think it is an excellent one. In connection with Dr H. W. Scott, Jr., Dr Byron Olson and others, we have been investigating and are continuing an investigation of the effect of congestion brought about by various types of constricting agents on the pulmonary circulation in regard to the therapy of tuberculosis. So far that has been an inconclusive study.

The most important thing to our mind about the present investigation is not the fact that the rate of survival or death can be altered, rather our interest lies in the mechanism by which compensation is brought about. As Dr Burke mentioned, one would think from the gross and histologic appearance that these lungs might be functional, but it is obvious when one tries to employ such a lung as a ventilatory agent that it is far from adequate functionally.

FURTHER DISCUSSION OF THE RIGHT THORACIC APPROACH TO ESOPHAGEAL LESIONS

R K HOLLINGSWORTH, M D , MARTINSBURG, W VA

SINCE it has been demonstrated that the stomach can be brought to any desired height in the thorax, or even into the neck, for esophagogastrostomy, some controversy has arisen over the virtues of the right- versus the left-sided approach. This applies more specifically to lesions of the upper and mid-thoracic esophagus. That lesions in the lower third are best handled through the left side is probably universally admitted. The ready access to the stomach through the left hemidiaphragm, without further external incision, is appealing. But in the higher lesions this advantage must be weighed against the greater difficulty encountered in removing them from behind the aortic arch and from the upper mediastinum. As Lewis¹ has pointed out, on the right, the azygos vein is the only impeding structure, and the aortic arch and the left subclavian artery act as a bolster against the left pleura during dissection. Furthermore, many thoracic lesions involve the azygos and are difficult to handle from the left. Just a few short years ago, before the days of thoracic esophagogastrostomy, the right side was the generally accepted one for the Thorek procedure. But this technically easier operation was overshadowed by the ease with which the stomach can be handled from the left, and this side became the one of choice.

In 1946 Lewis¹ reported a staged procedure in which he first explored the abdomen and mobilized the stomach. At a subsequent operation, the right side of the thorax was explored and, if possible, the esophagus was resected. The previously mobilized stomach was then brought up into the chest through the esophageal hiatus, and esophagogastrostomy was performed. This procedure had two very obvious defects. First, a rather formidable major abdominal operation was performed with no assurance that the second stage would be possible. Second, delay between the first and second stages produced technical difficulties in the form of intra-abdominal adhesions involving the previously mobilized stomach. Kay,² in 1941, had encountered this same difficulty. Chamberlain³ and Pool⁴ counter the first objection by performing the anastomosis above the esophageal lesion regardless of whether or not it can be removed. The second objection is obviated by performing the two stages on the same day. Both can be overcome by the use of a right thoracotomy incision which gives a wide, beautiful exposure of the entire area to be attacked. More recently, Harrison and Pickett⁵ have described a multiple incision approach, exploring the chest first through the third right anterior interspace, freeing the esophagus and then proceeding to a left abdominal incision to mobilize the stomach. While this has the advantage, as pointed out

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by the authors, of saving time by having two operating teams working simultaneously, one on each incision, it seems to me that technically the removal of the esophagus through an anterior incision could be very difficult

In this communication, three cases using the right-sided approach are reported. Each presents a different problem from which certain lessons may be learned



Fig 1—Case 1 Preoperative esophagogram

CASE REPORTS

CASE 1—S. T., No. 10 579, a 54 year old white man, was admitted to this hospital on Feb. 24, 1949. He was admitted to the Neurological Service for a nervous disorder. His history indicated that for the past two or three months he had suffered loss of weight and appetite and some epigastric distress following meals. This distress simulated gastric or duodenal ulcer, and in the course of routine barium studies a lesion of the midportion of the esophagus was seen (Fig. 1). On March 11, 1949, an esophagoscopy was done and biopsy made. This revealed a squamous cell carcinoma. Physical examination and all laboratory studies were within normal limits.

Operation was performed on March 22, 1949. The multiple approach as described by Lewis was done: a left subcostal incision for the abdominal part and through the bed of the resected sixth rib for the thoracic part. The carcinoma had invaded completely through the wall of the esophagus (Fig. 2) and removal was difficult, in fact it was im-

possible to remove all of the growth from the mediastinum, making the procedure entirely a palliative one. The operation was well tolerated, and the patient was returned to his room in good condition. Two months after operation this patient was sent to Walter Reed Hospital where he received deep x ray therapy, particularly centered over the area where the tumor was known to have been left. At autopsy no evidence could be found that this therapy had been of any benefit.



Fig. 2—Case 1. Surgical specimen. Note invasion of growth through entire wall.

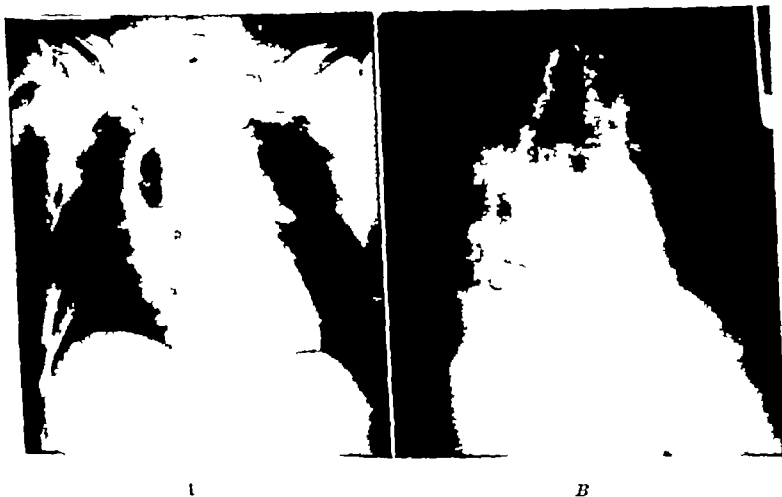


Fig. 3 (Cont.)—A. Portable film immediately postoperative. B. Later film showing marked dilatation of stomach with retained food.

Comment—Immediate postoperative recovery was without incident until the patient began to take food. It was then discovered that while swallowing was normal the stomach was not emptying, and it became markedly distended. This was thought to be due to a pylorospasm following the vagotomy incident to the resection. This gave considerable difficulty until finally, by the use of a liquid diet, nightly aspirations of the stomach and the use of Doryl, the

stomach recovered sufficient tone to force its contents through the pylorus. Emptying remained slow, and the stomach remained dilated (Fig 3). However, this did not seem to affect the patient's breathing. Further consideration of this complication led to the belief that perhaps the hiatus was too small. Additional comments on this will be made later.

This man lived seven months, after which he died of extensive metastases. At autopsy there was no evidence of recurrence at the site of the anastomosis. The stomach was markedly dilated and the stoma through the esophageal hiatus admitted two fingers but was filled with thick folds of gastric mucosa.



Fig 4—Case 2. Preoperative esophagogram showing stricture.

The site of the original lesion in the mediastinum showed extension of the growth in spite of maximum doses of deep x-ray therapy. Scattered metastatic nodules were seen in the mediastinum, stomach wall, on the right pleura, in the liver, and in the spleen.

Technically, the multiple incision approach was entirely satisfactory in this case, and I feel that mobilization of the esophagus was easier than it would have been from the left side. Throughout the operation the patient's condition was good. It did not appear that the slight increase in time required to open and close the abdomen added to the risk, and mobilization of the

stomach is the same whether done through the abdominal incision or through the left hemidiaphragm. Some time is lost in changing position, i.e. preparing and draping, and this is a disadvantage.

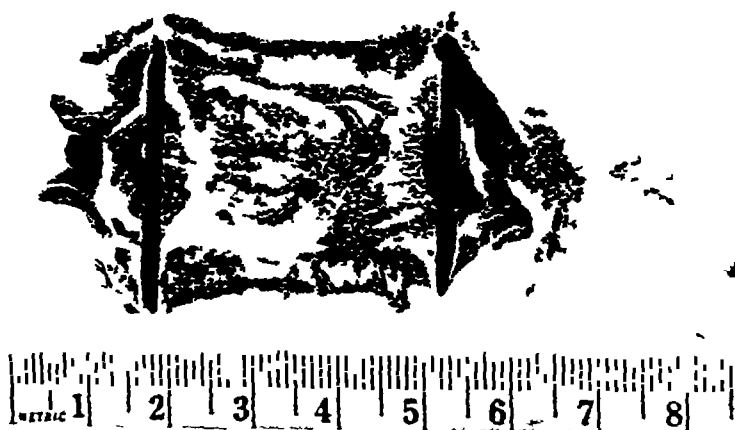


Fig 5—Case 2. Surgical specimen showing extent of stricture.



Fig 6—Case 2. This shows the level of the anastomosis and the postoperative dilatation of the stomach.

CASE 2—A I, No 8 233, a 49 year old white man, was admitted to this hospital on Nov. 8, 1948, with the chief complaint of difficulty in swallowing. Three weeks previous to this date he had picked up a bottle which he thought contained whisky, and swallowed its contents. He very promptly determined his error (it was lye) and drank large quantities of milk, chocolate milk, and water, finally vomiting. While remaining on a liquid diet, he continued to vomit daily, usually with the production of some blood. Nine days later he had severe substernal pain, and vomited a large quantity of blood. At the time of his admission here he was unable to swallow anything. Briefly, his treatment from this time until he was referred for surgery on April 25, 1949, consisted of a series of dilations. The stricture could be dilated up to F26, but the relief obtained was of short duration. Physical examination and all laboratory studies were within normal limits.

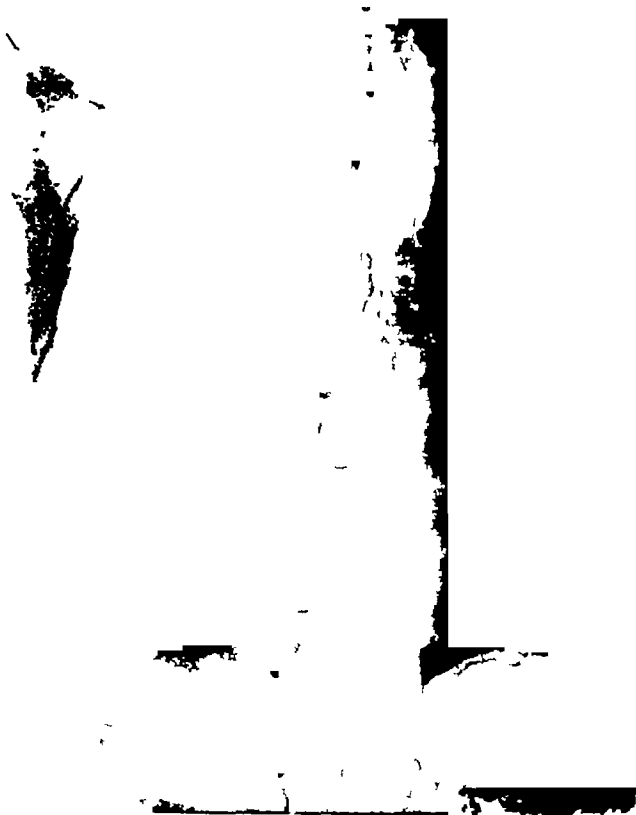


Fig 7—Case 3 Preoperative esophagogram

Operation was performed on May 4, 1949. We were not sure of the exact length of the stricture, but it was hoped that the involved portion could be resected and a direct esophageal anastomosis made. For this reason, a right sided approach was selected. When the stricture was found to be too long (Fig 5), complete mobilization of the lower segment of the esophagus, through the hiatus, was done. By drawing up a small amount of the stomach through the hiatus the ends of the esophagus could be approximated. However, it was feared that the blood supply of the lower segment might have been too involved and when it was found that an adequate amount of stomach could be brought up to reach the upper segment esophago-gastrostomy was felt to be the safest procedure.

The patient withstood the operation well, and made an uneventful recovery up to the time of taking food. As in Case 1, the stomach was slow to empty, but this man had much less trouble even though the stomach has remained dilated (Fig 6). When seen recently he had no complaints.

Comment—This case demonstrates the great mobility of the stomach without the division or any of its attachments. The fundus was easily brought up through the hiatus to the right midchest (Fig 6), and the anastomosis made without undue tension.

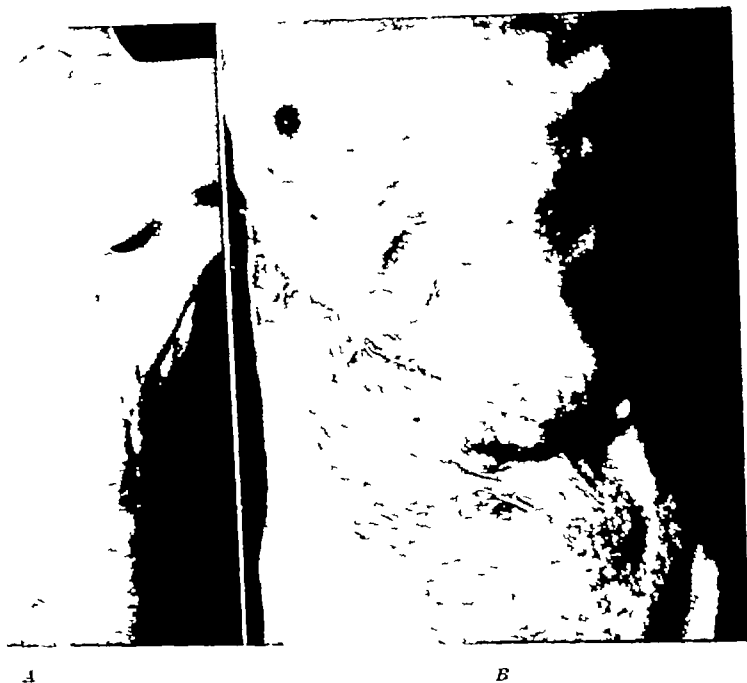


FIG. 8—Case 3. Showing *A* thoracic and *B* the abdominal portion of the combined thoraco-abdominal incision.

Here again the bad effects of the small hiatus crowded with stomach is demonstrated.

CASE 3—W. F., No. 12 479, a 57 year old Negro man, was admitted on July 31, 1950 with the chief complaint of difficulty in swallowing. The trouble had begun about two months previously when he said that he felt a 'sort of lump in his throat'. However, he was able to swallow all right until about three weeks previously when he began to have difficulty with solid foods. The condition progressed rather rapidly from this time until at the time of admission only liquids could be swallowed. There had been a thirty pound weight loss in the previous two months and there had been increasing ease of fatigue.

The past history and family history were noncontributory. His occupation was that of a gardener. Habits were good, smoking being moderate and drinking only a small amount of beer.

The physical examination contributed nothing to the diagnosis. While he was thin and showed evidence of recent weight loss, his general appearance was fair. Temperature and pulse were normal, blood pressure 100/70, weight 17 pounds, usual weight 165 to 170 pounds. All laboratory studies were within normal limits. Roentgenogram (Fig. 7) showed a filling defect in the esophagus extending from about D5 to D6.

At esophagoscopy on Aug 5, 1950, a fungating mass was encountered at about 25 cm. from the front teeth. A biopsy from this mass was reported as squamous cell carcinoma.

Operation was performed on Aug 10, 1950. A right thoraco abdominal approach was selected. Only such details as are pertinent to this approach will be described. The rest was routine procedure. The patient was placed on his left side, with the body at an angle of about 65 or 70 degrees and the chest and upper abdomen prepared and draped. The incision (Fig 8) was made along the seventh rib from the spinal muscles posteriorly to the costochondral junction, and the chest cavity entered through the bed of the resected seventh rib. When the esophagus had been mobilized from the thoracic inlet to the hiatus, the rib spreader was removed, the wound covered with sterile towels, and the table rotated toward the right until the abdomen was nearly level. The thoracic portion of the

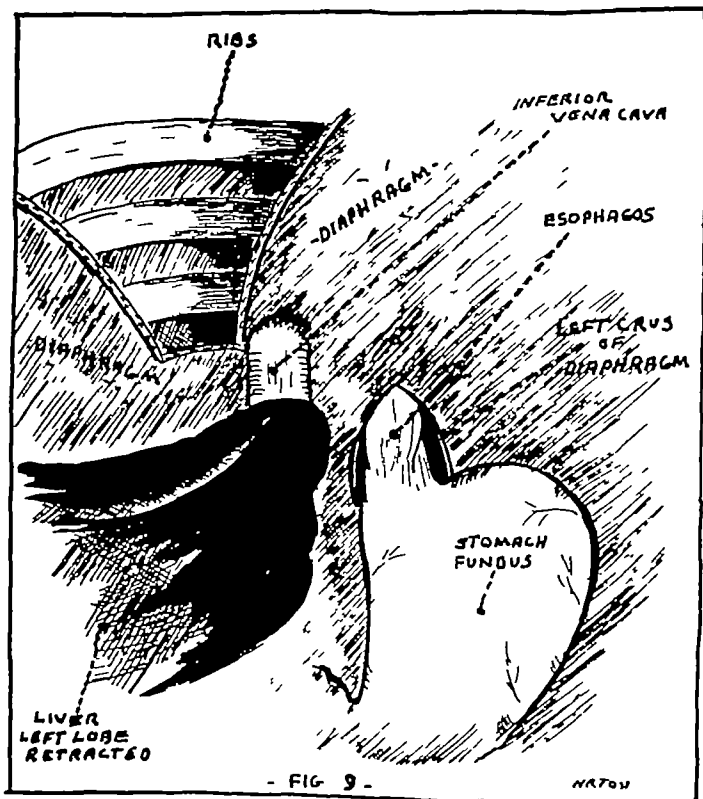


Fig 9—Normal relationship between esophagus and inferior vena cava.

incision was then continued across the costal margin to the left side of the abdomen. The hepatic ligament was then divided, mobilizing the left lobe of the liver which was retracted to the right, exposing the entire stomach. The spleen was somewhat enlarged and adherent to the greater curvature of the stomach. It was friable and, while being freed, it was torn and removed. The mobilization of the stomach was routine. With the left lobe of the liver retracted to the right, the entire gastrohepatic ligament was exposed and accessible. On drawing the stomach up into the chest, the cause of the difficulty in emptying the stomach encountered in Case 1, and to a less extent in Case 2, was well demonstrated. The esophageal hiatus is behind and slightly to the left of the inferior vena cava. The size of this hiatus is of relative unimportance. That it will vary in size has been

demonstrated by Harrington.⁶ Furthermore, if a larger hiatus is needed the right crus can be divided, giving more room in a transverse direction (Fig 9). The esophagus in its passage through the lower posterior mediastinum remains to the left of the inferior vena cava. But, to bring the stomach into the right chest, it (the stomach) must pass between the vena cava and the body of the vertebrae (Fig 10). Palpating through the esophageal hiatus from below one is impressed by the narrow space which exists between the vein and the vertebrae. That there can be serious interference with the emptying of the stomach is quite evident, particularly in view of the loss of tone occasioned by the division of both vagus nerves. The degree of obstruction, other than that caused by section of the vagi, depends on three factors. One, the size of the stomach, two, the thickness of the stomach wall, and three, the size of this space which may vary somewhat indi-

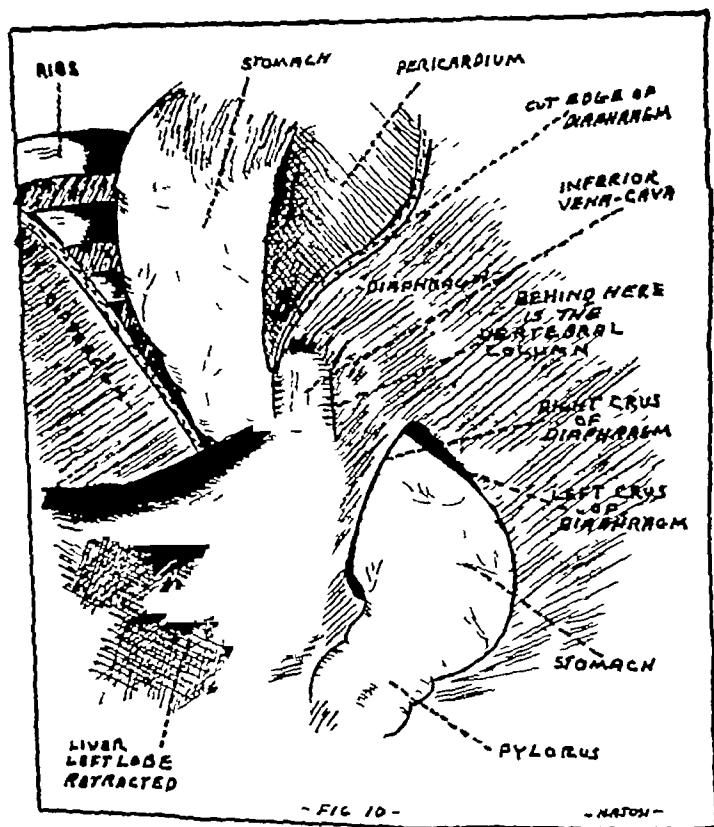


Fig. 10.—Stomach must pass between inferior vena cava and body of vertebrae to reach right chest.

vidually as do all anatomical structures and spaces. The remedy was quite evident: the transplantation of the stomach into the chest anterior to the inferior vena cava. The diaphragm was therefore split down to the vena cava. The esophagus was divided at the cardia and the stump inverted into the stomach. The normal esophageal hiatus was then closed with two interrupted sutures. The stomach could now be brought up into the chest just anterior to the vena cava and the diaphragm sutured to its wall, forming a new adequate hiatus. In addition to relieving the obstruction possibility, this maneuver added several centimeters to the length of the stomach available for high thoracic or cervical anastomosis. Closure of the abdomen, the performing of the anastomosis, and closure of

the chest were routine. The patient withstood the procedure well, and made a good post operative recovery. Sips of water were allowed on the second day, and soft food was easily swallowed by the end of the first week. There has been no difficulty with emptying of the stomach (Fig 11). This patient is not believed to be cured, as the mediastinum at about the level of the azygos vein was found to be infiltrated with the cancer, and it is very doubtful that it was all removed. But he is able to eat and has been comfortable these five months since the operation.



Fig. 11—Case 3. Three months postoperatively. Note tubelike character of stomach.

Comment—The combined thoraco abdominal approach was found to be a very satisfactory method of attacking a high or midthoracic esophageal lesion. Because of the mediastinal involvement, I am convinced that esophageal removal was decidedly easier than it would have been from the left side. Exposure of the entire upper abdomen is excellent, and the stomach is easily mobilized from the hiatus to the pylorus.

In spite of the technically easier mobilization of the esophagus from the right side I must admit that delayed emptying of the stomach encountered in Cases 1 and 2 had left me somewhat discouraged. With the combined ap-

proach, the cause of the difficulty was recognized and easily remedied. Fig 11 shows the present condition of the stomach. There is no dilation, in fact, it has now assumed a tubelike character.

It must be conceded that this operation is of greater magnitude than the left thoracic approach, with entrance to the abdomen through the diaphragm. However, I believe that in mid or high thoracic lesions the greater ease of mobilization of both the esophagus and the stomach compensates for the more extensive incision. Admittedly, one case is scarcely sufficient properly to evaluate a procedure, but it is enough to demonstrate and evaluate its technical aspects.

SUMMARY

The approach to lesions of the esophagus through the right chest is briefly discussed.

Three cases are reported, each presenting a different problem and from which certain lessons may be learned.

In two cases the bad effects of bringing the stomach through a small hiatus is demonstrated.

In one the great mobility of the stomach without dividing its attachments is demonstrated.

One case is reported, using a right combined thoraco abdominal incision. If the right approach is to be used, it is believed that this is the most satisfactory incision.

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Announcement

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The 33rd annual meeting of the American Association for Thoracic Surgery will be held March 27, 28, and 30, 1953, in San Francisco, Calif. Headquarters will be at the Hotel Fairmont. Suitable accommodations also have been reserved at near by hotels, the Mark Hopkins, the Huntington and the Sir Francis Drake.

Requests for Hotel Reservations

These should be mailed to Dr. H. Brodie Stephens, Chairman of Hotel Committee, American Association for Thoracic Surgery, Room 200, 61 Grove St., San Francisco 2, Calif. The following information must be included: (1) two choices of hotels, (2) type of accommodation desired—single, double, twin, room or suite, (3) name of other room occupants, if any, whether wife or another doctor, (4) date and approximate hour of arrival, (5) date of departure, (6) return address.

Abstracts for Papers

Abstracts for the presentation of papers at the 1953 meeting must be received on or before Dec. 1, 1952, otherwise they will not be considered by the Program Committee. *Five, repeat, five* copies of each abstract should be sent to the Secretary of the Association, Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif. They should not be sent to the Editor.

The abstracts should contain from 200 to 250 words, and should accurately reflect the contents of the completed paper.

Applications for Membership

Completed membership applications must reach Dr. Edward M. Kent, Chairman of the Membership Committee, 1107 Clark Bldg., Pittsburgh 22, Pa., on or before Dec. 1, 1952, in order to be acted upon at the annual meeting in San Francisco.

All correspondence regarding membership, including requests for membership applications, should be addressed to Dr. Kent.

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Original Communications

LOCALIZED FIBROUS MESOTHELIOMA OF THE PLEURA

O THERON CLAGETT, M D, * JOHN R McDONALD, M D ** (BY INVITATION), AND
HERBERT W SCHMIDT, M D ***
ROCHESTER, MINN

DURING the past decade we have become increasingly conscious of a small group of circumscribed tumors situated in the thorax. They often are intimately associated with the pleura, but less commonly occur in the periphery of the lung and occasionally in the mediastinum. This group of tumors has not readily fallen into the more commonly accepted classification of intrathoracic tumors. It became apparent after studying these tumors that they are tumors sui generis, which because of their peculiar clinical course are of particular interest to the thoracic surgeon.

Klemperer and Rabin¹ said, "Although primary pleural neoplasms are classed among the rarer types of tumors, their importance has been recognized in a rather extensive literature." According to Saccone and Coblenz² (1943), more than 200 authors had discussed and offered opinions regarding these tumors and had described the perplexities of their clinical, roentgenologic and pathologic features. Some evidence of the confusion that occurred in the literature is Krumbein's³ finding that thirty different names had been used to describe these tumors. In more recent years, some order has come out of the chaos that has existed. Notable contributions toward clarification have been made by Klemperer and Rabin¹ and by Stout and Murray.⁴ It now seems reasonable to divide tumors arising from the pleura into two distinct groups. The first but less common group consists of localized tumors resembling fibromas or fibrosarcomas, and the second group includes tumors of a diffuse nature involving the entire pleura, both parietal and visceral. Our experience is in

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Division of Surgery
Division of Surgical Pathology
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general agreement with that of Klemperer and Rabin who said, "Localized tumors may involve the parietal or visceral pleura. They comprise many histologic types and benign and malignant forms. The entire group is possessed of a common characteristic. They originate from the tissue beneath the superficial lining cells of the pleura in contradistinction to the diffuse form of pleural tumors which arise from the surface lining." These two groups of tumors are entirely different as regards their gross and microscopic characteristics, their clinical manifestations and course, and their treatment and prognosis. Much of the confusion that has existed is due to the fact that these 2 groups of tumors of pleural origin have been considered as a single entity in much of the literature whereas they actually have nothing in common except their supposed origin in the pleura. As a result of our experience with localized fibrous mesotheliomas of the pleura, we believe that these tumors constitute a definite disease entity both pathologically and clinically.

It has been difficult to select a proper name for the group of tumors from the thirty terms that have been applied in the past. Since both localized and diffuse pleural tumors have a mesothelial origin, and since the term "mesothelioma" recently has gained acceptance, it seems wise to accept this term. However, in order to distinguish these groups of tumors sharply we shall name the tumors under discussion "localized fibrous mesotheliomas," and the other group, "diffuse malignant mesotheliomas."

There are no accurate figures in the literature to indicate the true incidence of localized fibrous mesotheliomas. According to Ramstrom and Hellsten,⁵ Lichenstem⁶ was able to collect reports of only 8 cases of tumors of this type from medical literature up to 1931. These authors found reports of 3 more cases in the literature between 1931 and 1951, and added reports of 3 new cases, making a total of only 14 cases reported up until 1951. Undoubtedly reports of many cases were overlooked in the confused literature on this subject, however, because we have been able to collect a series of 24 cases of tumors of this type in which operation was performed at the Mayo Clinic. There were 14 males and 10 females in our series, a slightly different ratio from the 2:1 ratio mentioned in some of the reports in the literature. The age of the patients in our series ranged from 21 to 70 years, and the average age was 49.3 years.

The association of pathologic changes in bones and joints with intrathoracic tumors and diseases has been noted frequently since it was described independently by Bamberger and Marie⁷ in 1890. Van Hazel⁸ presented a paper on this subject before the American Association for Thoracic Surgery in 1939 and reported a series of 7 cases of tumor of the lung in which the initial symptoms had been changes in the joints. In 5 of the 7 cases, the tumor was malignant, in the 2 remaining cases, it was benign. Although articular metastases do occur in some cases of bronchogenic carcinoma, they are not common. In our series of more than 200 cases of carcinoma of the lung, we found only 4 in which the articular metastases were the first to be noticed.

erable to joints. It was therefore of considerable interest to note that in 16 of the 24 cases of localized fibrous mesothelioma of the pleura there were symptoms or physical findings, or both, referable to joints. In many of the cases, these symptoms were primarily responsible for bringing the patients to a physician. Articular symptoms had been present for from four months to sixteen years. In 9 cases, these symptoms had been present for more than one year. The most frequent sites of articular involvement in order of decreasing frequency were the hands, ankles, shoulders, wrists, elbows, and knees. In some cases, the patients were almost entirely disabled by the articular manifestations. When the articular reactions were of short duration and unaccompanied by clubbing of fingers and toes, it was impossible even for rheumatologists to distinguish clinically the arthritis from rheumatoid arthritis occurring in patients without thoracic tumors. Clubbing of the finger tips or toes or both (Fig 1), sufficient to attract the attention of the physician, occurred in eleven cases, and minor degrees of clubbing may well have been overlooked in some patients who were seen before we became fully aware of the frequent association of this finding with pleural lesions of this type. In 7

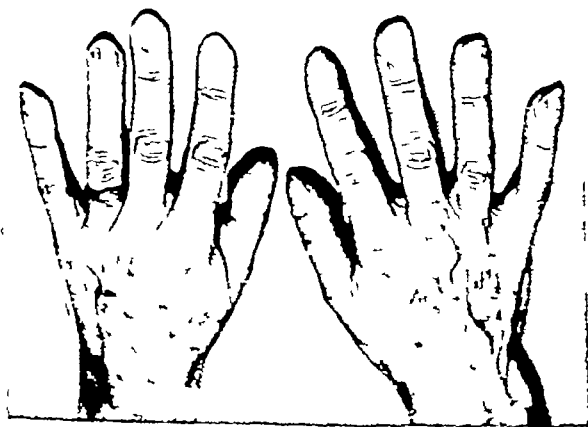


Fig. 1—Typical clubbing of finger tips in case of fibrous mesothelioma of the pleura.

cases there was a history of recurrent attacks of chills or chilly sensations and fever of unknown cause, having occurred before the intrathoracic lesion was discovered. Fever was found generally to occur only in patients with symptoms referable to their swollen painful joints. It would appear that chills and fever may well be considered a part of the clinical picture in some cases of localized fibrous tumors arising from the pleura.

Seven of the patients with large tumors complained of dyspnea but it was not an important symptom. Loss of weight was rarely observed. Thoracic pain was common in many cases. In a number of cases the pleural lesions and the intrathoracic lesions were discovered at the same time. In some cases the pleural lesions were discovered first and the intrathoracic lesions were discovered later.

The physical findings other than clubbing of the fingers and swollen painful joints were those that would be expected in any case of intrathoracic tumor of similar size. No abnormal physical findings were noted in cases in which the tumors were small.



Fig 2—*a* Anteroposterior roentgenogram showing area of increased density in lower portion of right lung *b* lateral roentgenogram showing an ovoid interlobar tumor *c*, fibrous mesothelioma of pleura responsible for roentgenographic changes shown in *a* and *b*

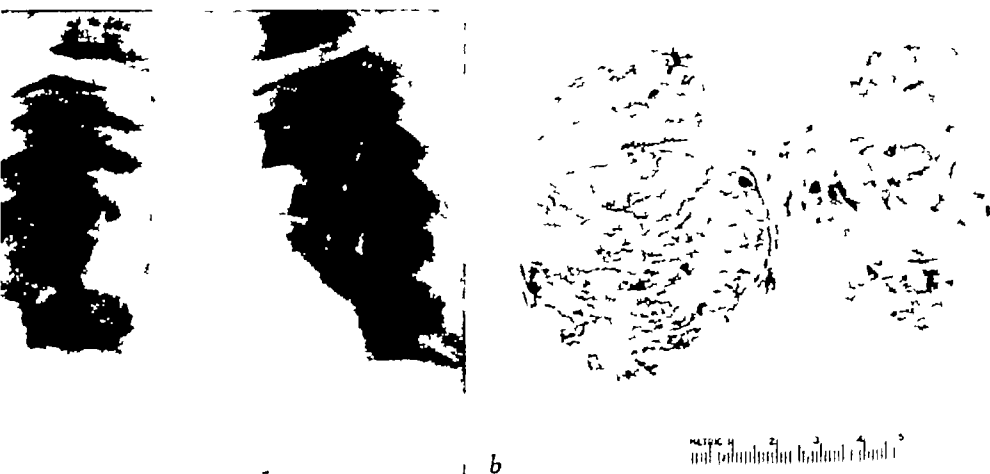


Fig 3—*a* Anteroposterior roentgenogram showing rounded shadow of increased density in lower part of right side of thorax *b* fibrous mesothelioma of pleura responsible for roentgenographic changes in *a*

DIAGNOSIS

It must be admitted that a definite diagnosis of fibrous pleural tumor was not made preoperatively in any of the cases in this series. However, the possible presence of this lesion was considered in a few cases. In most cases exploratory

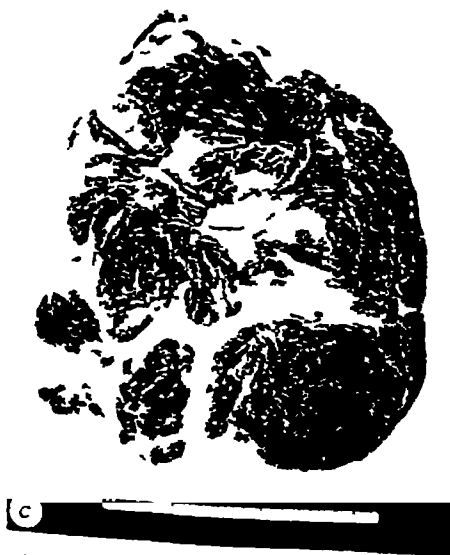
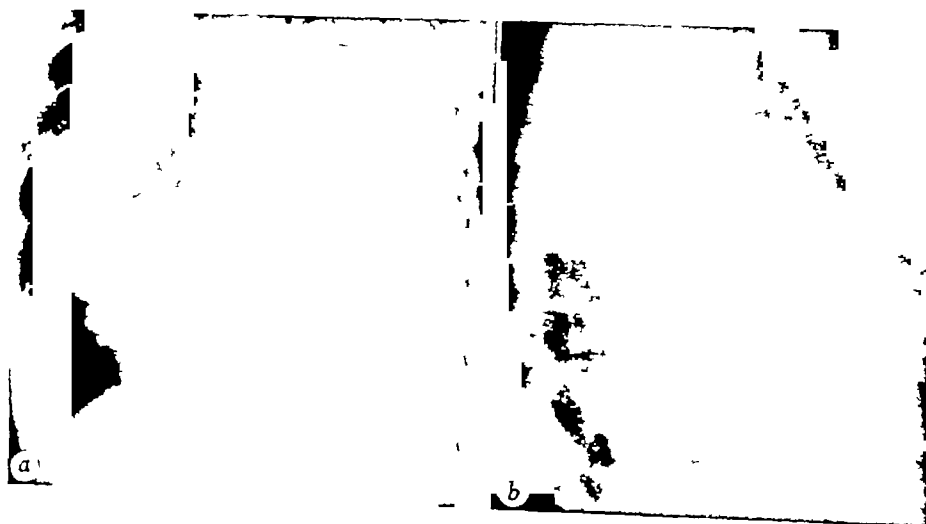


Fig. 1—*a* Anteroposterior roentgenogram showing shadow of huge tumor filling the left side of the thorax and causing deviation of the mediastinum to the right. Some calcification is evident in the periphery of the tumor. *b* Lateral roentgenogram showing huge lobulated tumor filling the left side of the thorax. *c* Mesothelioma of the pleura responsible for the roentgenographic changes shown in *a* and *b*. The tumor weighed 3450 grams.

thoracotomy was advised because of a preoperative diagnosis of indeterminate intrathoracic tumor. Bronchoscopy was performed in a few instances but, as would be expected considering the pleural origin of these tumors, it did not contribute anything to the diagnosis except to exclude the presence of a lesion in the major bronchi.

Thorough roentgenographic studies in each case disclosed the presence of an intrathoracic lesion and provided important information regarding the site and size of the tumor. However, roentgenographic examination did not disclose any features that would permit one to distinguish these tumors from a variety of other intrathoracic tumors. Although roentgenographic examination disclosed the presence of an intrathoracic tumor in all of the cases in this series, the roentgenographic findings did not indicate definitely that the tumor was of pleural origin in any of the cases (Figs 2, 3 and 4).

Although a correct preoperative diagnosis was not made either clinically or roentgenologically in this series of cases, it would seem that if we profit from the knowledge gained from our review of them we should be able to diagnose at least some of these tumors preoperatively in the future. Although other intrathoracic lesions can produce articular symptoms and clubbing of the fingers in some cases, we know of no other intrathoracic tumor in which a clinical history of chills, fever, migratory swelling and pain in the joints, and clubbing of fingers occur so commonly. A clinical history of this type obtained in a case of circumscribed intrathoracic tumor should make one consider the possibility of localized fibrous mesothelioma.

SURGICAL CONSIDERATION

The tumors removed at operation varied in size from 4 cm. to 36 cm. in diameter and from 50 to 5,000 grams in weight. In many cases in which the tumors were large, it was impossible to determine the exact site of origin of the tumor. As accurately as could be determined, the tumor arose from visceral pleura in 18 cases and from parietal pleura in 6 cases. Thirteen of the tumors arising in the visceral pleura were situated in the right side of the thorax and five in the left. Two tumors which originated in the parietal pleura were situated extrapleurally, two apparently arose from the mediastinal pleura, and two arose from the pleura of the lateral wall of the thorax but were situated intrapleurally. Grossly and microscopically, all of these tumors, regardless of their situation or site of origin, seemed identical.

It should be noted that pleural effusion was present in 4 of the 24 cases. In 3 of these cases, the presence of pleural effusion was established preoperatively by thoracentesis, in the fourth case, a few hundred cubic centimeters of clear fluid that had not been suspected preoperatively were found at operation. In one instance, bloody pleural fluid was aspirated on several occasions preoperatively. In all cases in which pleural effusion was found, the tumor is removed successfully. Hawthorne and Froese¹⁰ have reported removal of a fibrous pleural tumor which was associated with a pleural effusion.

In 18 cases, the tumors were removed by excision without pulmonary resection. Some of the smaller tumors were attached to the lung by a small vascular pedicle (Fig 5), and only a ligature or a few sutures were necessary to accomplish complete removal of the tumor and repair of the lung from which the tumor had originated. Large tumors often were attached to the lung and thoracic wall by vascular adhesions. However, except in the cases of very large tumors, the operations usually were not difficult. One or more lobes of the lung were removed together with the tumor in 6 cases. Pneumonectomy was performed in only one case. The tumor, in that case, was embedded in the fissures between the lobes close to the hilus of the lung. Since biopsy indicated that the tumor was a fibrosarcoma, pneumonectomy seemed indicated.

There were two postoperative deaths in this series of 24 cases. One occurred the day of operation which had included removal of the tumor together with the left upper lobe. This operation was performed in 1931 long before thoracic operations of this magnitude were commonly performed. The result in this case is not very surprising. The other death occurred one week after operation. There were no major postoperative surgical complications in this case; the patient dying of hypertensive heart failure.



FIG. 5—Fibrous mesothelioma of the pleura attached to lung by a pedicle.

As accurately as we can determine from our follow up investigations, there has been local recurrence of the tumor in 4 cases and so far as we can determine, with one possible exception, none of the tumors has metastasized to other parts of the body. In 2 cases the tumor recurred within six months. These two recurrent tumors were treated with deep roentgen therapy, but the patients died within two years. In the third case the tumor recurred after eight years and the patient died two years later. In the fourth case the recurrent tumor was noted four years after operation, and the patient died a year later.

One patient who was 70 years old at the time of operation died one year later. We have not been able to learn the cause of this patient's death. All of the other patients were well and free of symptoms at the time of a recent follow-up study.



Fig. 6—Section of fibrous mesothelioma of pleura showing variation in cellular pattern. Some areas are relatively acellular and contain dense bundles of collagen while others are very cellular (Hematoxylin and eosin stain $\times 50$).

GROSS FEATURES

In all of the cases in this series the tumors were encapsulated. In the majority of cases, gross examination revealed definite lobulation which was particularly prominent on the surface of the tumor. Bosses were present on the surface of the tumor. On section, some of the tumors had an appearance which suggested placental tissue, and cystic formation occasionally was evident. Calcium was present in one tumor (Case 21), necrosis and hemorrhage were present in only one case (Case 22). The latter was the only case in which histologic examination disclosed undeniable evidence of malignancy. The weight of the tumors varied greatly (Table I). Three of these tumors were very large and weighed from 8 to 10 pounds (3.6 to 4.5 kg). The site of the tumor in the thorax varied. In 13 of the 24 cases the tumor was pedunculated, it was attached to either the visceral or parietal pleura and projected into the pleural cavity. Pleural fluid was present in 4 of the 13 cases. Six of the tumors were embedded in the lung close to the visceral pleura. Other tumors appeared to arise primarily as mediastinal enlargements. A few were attached to the pleura with a broad pedicle and projected a short distance into the pleural cavity.

HISTOLOGIC FEATURES

All of the tumors were composed of spindle cells which produced collagen intercellularly. Some portions of the tumors were relatively acellular, and the cells that were present in these portions were slender spindle cells. A comparatively large number of spindle cells were present in other portions of

the tumors, and mitotic figures occasionally could be seen (Fig 6). In these relatively cellular portions, the spindle cells were more plump than those in the relatively acellular portions. In the latter portions, fingerlike projections or dense bundles of collagen were arranged in an interlacing manner. In the relatively cellular portions, the collagen was much finer and frequently appeared only as precollagen in sections which were stained with Gomori's reticulum stain. These fingerlike projections of bundles of collagen were a prominent feature of the tumors in this series of cases (Fig 7). They probably account for the fact that a fibrous mesothelioma occasionally has been mistaken for a neurofibroma. These projections, however, are not a prominent feature of fibromas.

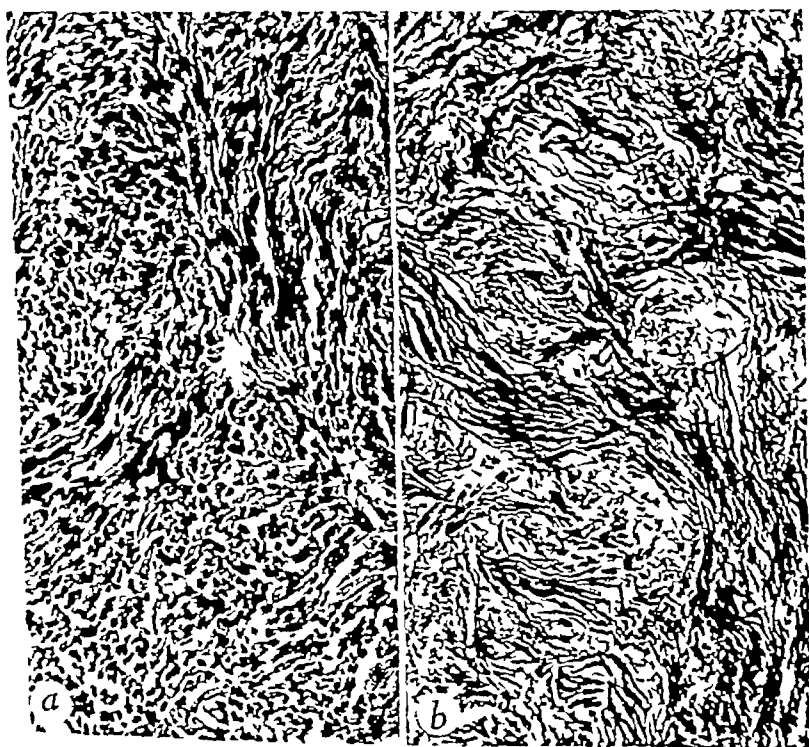


Fig 7.—Sections of relatively acellular portion of tumor showing dense bundles of collagen. a, section stained with Masson's trichrome; b, section stained with Gomori's reticulum stain.

of pleura showing (X145) b, section

Mitotic figures were found in a small proportion of the tumors in this series of cases. They usually were situated in portions of the tumor which contained a relatively large number of cells. A large number of these figures were present in only 1 case (Case 22). In this case, the cells and nuclei were large and irregular, the latter were hyperchromatic, and the histologic picture of the entire tumor resembled that of a malignant tumor (Fig 8). The clinical course also suggested that the tumor was malignant, because it recurred locally a short time after its removal but definite evidence of metastasis was lacking.

TABLE I CLINICAL DATA IN 24 CASES OF LOCALIZED FIBROUS MESOTHELIOMA OF THE PLEURA

CASE	AGE (YEARS)	SEX	ARTICULAR PAIN AND SWELLING	DURATION OF ARTICULAR SYMPTOMS	CLUBBING OF FINGERS	KNOWN DURATION OF THORACIC LESION	WEIGHT OR DIAMETER OF THORACIC LESION	ORIGIN OF TUMOR	OPERATION	RESULT
1	32	F	+	9 mo		9 mo	130 Gm	Visceral pleura of middle lobe	Local excision	Well, Feb, 1952
2	19	M	+	9 mo		9 mo	9 Gm	Visceral pleura of lower lobe	Local excision	Well, Jan, 1952
3	53	F				3 yr	165 Gm	Parietal pleura	Local excision	Well, Feb, 1952
4	40	M	+	6 mo			4 cm	Visceral pleura of right lower lobe	Local excision	Well, Jan, 1952
5	71	F	+	15 yr		4 yr	180 Gm	Parietal pleura (extrapleural)	Local excision	Recurrent tumor excised six mo later, death 2 yr after excision of primary tumor
6	17	F	+	16 yr	+	1 wk	3,480 Gm	Visceral pleura of left lower lobe	Local excision	Well, Jan, 1952
7	36	M	+	4 yr	+	5 yr	4,050 Gm	Visceral pleura of middle and lower lobes right lung	Removal of middle and lower lobes of right lung	Well, Jan, 1952
8	54	M				2 mo	75 Gm	Visceral pleura of right lower lobe	Local excision	Well, Jan, 1952
9	48	M	+	4 mo	+		220 Gm	Visceral pleura of left lower lobe	Local excision	Well, Jan, 1952
10	61	F	+	6 mo	+	2 wk	860 Gm	Visceral pleura of right upper lobe	Local excision	Well, Jan, 1952
11	58	M	+	28 yr*	+		140 Gm	Visceral pleura of right middle lobe	Local excision	Well, Jan, 1952
12	44	M	+	1 yr	+		4,972 Gm	Visceral pleura of right lower lobe	Local excision	Recurrence, death 5 yr after operation
13	59	F	+	4 yr	+		2,865 Gm	Visceral pleura of right lower lobe	Local excision	Well, Jan, 1952
14	55	M	+	15 yr			180 Gm	Mediastinum	Local excision	Well, 1 yr after operation
15	21	M				6 mo	55 cm	Beneath visceral pleura	Pneumonectomy	Well, Jan, 1952

In 1 case (Case 5), the clinical course did suggest the presence of embolic metastasis. In this case, a recurrent tumor was removed, and the patient died two years after removal of the primary tumor. At the time of the patient's death, multiple nodules were present beneath the skin. The histologic nature of these nodules was not determined. The histologic appearance of the tumor in this case did not differ from that of the tumors which followed a benign course clinically.



Fig. 8—Fibrous mesothelioma of pleura showing evidence of malignancy (hematoxylin and eosin stain, $\times 180$)

Necropsy was performed in 2 cases (Cases 16 and 18). In both of these cases, the necropsy was performed shortly after removal of the respective tumors. In neither case was any residual tumor found, nor was there any evidence of metastasis. Another almost constant finding in these 24 cases was the presence of spaces lined by spindle cells (Fig. 9). In 1 case (Case 20), these spaces were lined by columnar cells and had the appearance of actual glands. A number of these spaces contained coagulated proteinlike material which resembled secretion. The collagen bundles and cells appeared to be avoiding these spaces. Whether these spaces are true glands or attempts at the formation of coelomic spaces is conjectural. Mucoid degeneration was a feature of some of these tumors. It was, however, never as marked as one sometimes sees in fibrogenic tumors of the extremities.

COMMENT

We believe that our experience with this series of cases of localized fibrous mesothelioma should establish the fact that this tumor is a distinct pathologic and clinical entity which should be sharply distinguished from a diffuse malignant mesothelioma of pleural origin. Localized fibrous mesotheliomas of the pleura are of importance to the thoracic surgeon because these tumors are resectable. They are also of importance to the pathologist because he must learn to recognize the histologic patterns which they produce and to make a correct diagnosis of the tumor. Because of their cellularity it is very easy to label them as slowly growing malignant tumors and thus deter the surgeon from attempting to remove them. The fact that we have been able to collect, in the past twenty years, a series of 24 cases in which this type of tumor has been removed, suggests that these tumors occur more frequently than has been generally recognized. Undoubtedly, many reported cases of this type of tumor have been lost due to the confusing nomenclature that has been applied to them.

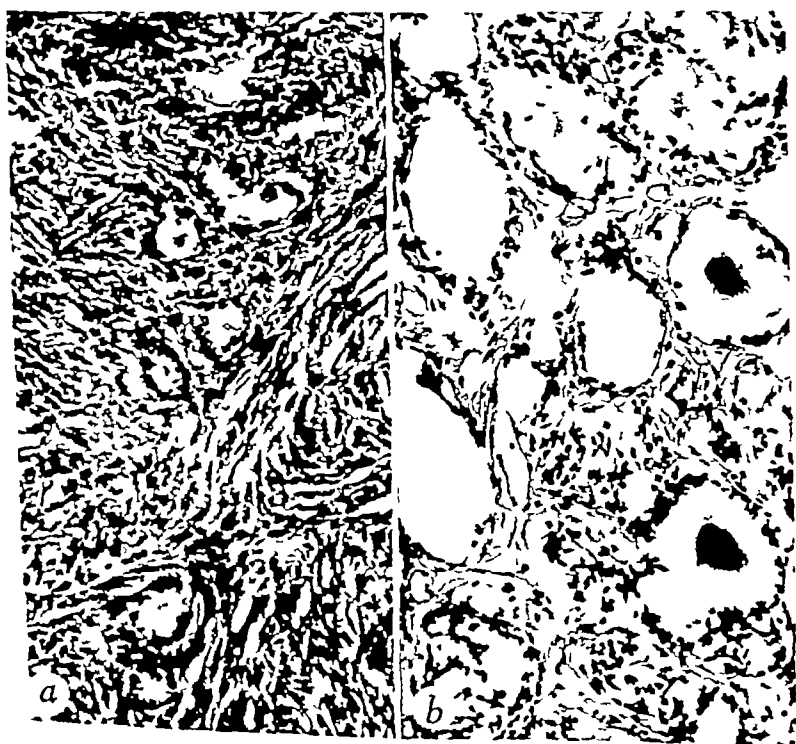


Fig. 2—Sections of fibrous mesothelioma of pleura showing spaces containing secretion. Sections stained with hematoxylin and eosin. *a* $\times 200$, *b*, $\times 145$.

It is of considerable interest that the clinical manifestations of these fibrous pleural tumors have been almost entirely extrathoracic. There can be little doubt that there is a direct association of some kind between these tumors and the attacks of chills or chilly sensations and fever, the swollen, painful, and

sometimes migratory involvement in the joints of the extremities, and the clubbing of the fingers or toes or both that occur in such a high percentage of patients with these tumors. Further evidence of this association is the prompt and complete relief of the attacks of fever and chills and the articular pain and swelling, and the gradual regression of the clubbing of the fingers and toes that have occurred following removal of the tumor in every case in which these signs and symptoms had existed preoperatively.

It is possible that the presence of clefts and glandlike spaces in these tumors has some relationship to a specific secretion by localized fibrous mesotheliomas which is responsible for the arthritic symptoms. It could be conjectured that such a secretion might be in the nature of hyaluronidase. This is suggested particularly in view of the fact that the hyaluronidase enzyme system is concerned in an ill-understood fashion in the production of certain types of arthritis. A study of the hyaluronic acid content of pleural and peritoneal fluid associated with a tumor involving the pleura and peritoneum was made by Meyer and Chaffee,¹¹ who found that on three separate occasions there was a constant yield of hyaluronic acid. We hope to make such a study in the future, but inasmuch as such work must be done on fresh tissue and because these tumors are rare, this phase of investigation will proceed very slowly. In one instance in our experience in which the tumor recurred four years after operation, there was simultaneous recurrence of joint symptoms and clubbing of the fingers. We are not able at this time to offer any reasonable explanation for this association. It is indeed frustrating to be confronted with such a definite relationship and not be able to understand it. We have absolutely no explanation for the production of chills and fever. Necrosis, which in other tumors is occasionally associated with the production of fever, was notoriously absent in all except 1 case.

Klemperer and Rabin¹ expressed the opinion that both benign and malignant forms of pleural mesothelioma exist. More cases must be studied over a longer time to permit one to come to any definite conclusion as to how many of these tumors are benign and how many are malignant, and what the histologic criteria for malignancy are. In one of the cases in this series histologic examination revealed unequivocal evidence of malignancy. In the other cases, the tumors, for the most part, appeared to be benign. However, in a series of cases of a new type of tumor, it is not scientifically feasible to determine which tumors are malignant and which are benign until a sufficient number of patients have been followed. We have not followed our patients long enough to determine this. To many people, the only unequivocal evidence of malignancy of an intrathoracic tumor would be embolic metastasis, and although there was clinical evidence of embolic metastasis to the skin in 1 case, this was not proved. The fact that four of the tumors recurred locally does not of necessity imply a malignant connotation. There was a recurrence of the tumor in 4 of the 22 cases in which the patients survived operation, and in all of the 4 cases the patients died of a recurrence of the tumor. However, the pathologic findings in these 4 cases were no different from those in the other cases in which there has been no recurrence.

Klemperer and Rabin¹ have reported 1 case of this type of tumor which was considered to be a low-grade fibrosarcoma when first removed. A recurrent tumor was removed subsequently. The recurrent tumor was highly malignant. This tumor ultimately metastasized to the other pleural space and the lung, and the patient died. Klemperer and Rabin postulated that repeated surgical interference was responsible for this malignant transformation. They stated that malignant transformation is observed commonly after incomplete removal of fibroblastic tissue.

The pleural effusion which was noted in 4 of our cases was not considered to be in any way associated with the malignancy of the tumor because it was not associated with implantation of the tumor in the pleural cavity. All of the tumors which were associated with pleural effusion were pedunculated tumors which were attached to either the visceral or parietal pleura by a narrow pedicle and appeared as a cauliflower-like mass projecting into the pleural cavity. It is possible that the production of fluid in such cases is comparable to that seen in the peritoneum in cases of Meig's syndrome in which various ovarian tumors, particularly fibroma with a narrow pedicle, give rise to fluid in some obscure fashion.

In view of the fact that thoracic surgeons everywhere accept the philosophy that indeterminate intrathoracic tumors must be explored, it seems likely that an increasing number of tumors of this type will be discovered. It is important that tumors of this type be kept in mind and recognized when they occur, so that unnecessarily radical operations will not be performed. It should rarely be necessary to destroy much if any lung tissue in removing these tumors. Failure to recognize the true pathologic nature of a few of the tumors in the series we have reported led to removal of normal lung tissue that could have been preserved. It is our belief that localized pleural mesotheliomas can be recognized from their distinctive histologic features alone, particularly if one is aware of the intrathoracic site of the tumors.

SUMMARY

This report is based on a series of 24 cases in which a localized fibrous mesothelioma was removed surgically. Of the 24 patients, 14 were men and 10 were women. The average age of the patients was 49.3 years. The clinical manifestations of these tumors are largely extrathoracic and are characterized in a high percentage of cases by attacks of chills, chilly sensations or fever, pain and sometimes migratory involvement of extremity, joints and clubbing of the fingers or toes or both. Prompt relief of these symptoms follows surgical removal of the tumor. There are no characteristic roentgenologic findings which permit these tumors to be distinguished from other intrathoracic tumors. Pleural effusion occurs in association with these tumors in some instances, but does not constitute a contraindication to successful surgical removal of the tumors.

Fibrous mesotheliomas are encapsulated and often are lobulated. Histologically they are fibrogenic tumors which contain numerous whorls of collagen

and the spindle cells. They contain focal collections of cells which appear to be malignant. The most significant finding is the presence of spaces or glandlike cavities.

Localized fibrous mesotheliomas may arise from any portion of the visceral or parietal pleura. The majority arise from the visceral pleura. They rarely are situated extrapleurally.

The only effective treatment of these tumors is complete surgical removal. Localized fibrous mesotheliomas must be sharply distinguished from diffuse malignant mesotheliomas.

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DISCUSSION

DR. H. C. MAIER, New York—I have been very much interested in this excellent presentation from the Mayo Clinic. The literature on pleural tumors was in a confused state until Dr. Arthur Purdy Stout greatly clarified the situation. He recognized the localized pleural mesothelioma on the basis of pathologic and clinical data and especially through tissue cultures done in collaboration with Dr. Margaret Murray. Previously, only the diffuse malignant pleural mesothelioma was generally recognized.

In many cases of pleural tumors that were originally designated as fibroma, leiomyoma, or sarcoma, careful search of the microscopic slide usually reveals areas with mesothelial lined spaces such as Dr. Clagett has just shown. (Slide.) In this slide such spaces are particularly prominent. These areas were not in lung tissue but in the part of the tumor that protruded into the pleural space. I mention this point because some years ago it was thought some of these spaces might be residual alveolar spaces. One would not expect such findings in a simple fibroma or leiomyoma, as many of these tumors have been called in the past.

(Slide.) Dr. Stout has kindly loaned me this slide which summarized the data collected in his laboratory of surgical pathology at Columbia University. He has studied 62 cases of mesothelioma of the pleura sent to him from many sources. Forty of these are benign, the type that Dr. Clagett has discussed, and 22 were malignant. Of 16 cases of pleural tumor that I have seen there has been a higher percentage of the malignant variety than in this collected group of Dr. Stout's. I agree with Dr. Clagett that one must differentiate the benign localized type from the malignant diffuse type. Only five of the 16 pleural tumors that I have seen have been of this localized benign type.

I would like to show two instances of other types of pleural tumor. (Slide.) This is a pleural hemangiopericytoma in which the pulmonary osteoarthropathy was very marked in a young female.

The next case is not one of the localized type which Dr Clagett discussed, but is a diffuse malignant lymphoendothelioma of the pleura. (Slide) This patient had chylous pleural fluid. The lateral recumbent film of the same patient shows the mass which was present on the pleural surface. Before her death this patient developed chyle in the peritoneal cavity. These rare cases illustrate that one can have other types of tumors develop from the pleural tissues in addition to the localized benign and diffuse malignant mesotheliomas.

DR. WILLIAM B. CONDON, Denver—I think studies such as these presented so ably by Dr Clagett certainly increase our knowledge of intrathoracic disease. Such studies are particularly valuable in the prognosis we can offer the patient and his relatives, and to corroborate Dr Clagett's studies I would like to present one recent case which came to our attention.

(Slide) This is a 66-year old woman who had been treated for arthritis for some years, of moderately severe type, whose chest symptoms were referable to a space-occupying lesion in the left thorax. (Slide) The lateral view shows a rather large tumor lying in the interlobar fissure which at the time of surgery presented a picture somewhat similar to this.

(Slide) With the exception of a very few avascular adhesions to the diaphragm, the tumor lay free in the chest cavity except for a broad pedicle which sprang from the dorsal division of the left lower lobe. This pedicle contained numerous large blood vessels. The tumor was easily removed by merely placing a clamp across the pedicle.

(Slide) On the first examination of the microscopic sections, a diagnosis of a malignant, highly cellular fibrosarcoma was made. However, when reviewed by several pathologists, the diagnosis was changed to that of a mesothelioma, which in spite of its cellularity and the number of mitotic figures, was thought to be consistent with a diagnosis of a benign type of tumor. The patient has remained free of any clinical recurrence in the last eighteen months. Her arthritis has improved markedly since surgery.

SIR CLEMENT PRICE THOMAS, London.—I would like to say a few words on this topic, not that I have anything further to contribute to what Dr Clagett has said. We have had several cases of this type during the last few years, and the first case caught us on one foot. This was a man who had had symptoms since the age of 16. I was interested to hear Dr Clagett state that these people have symptoms referable to conditions outside the chest. The outstanding feature of course is the arthritis or the pulmonary osteoarthropathy from which they suffer. This man's initial symptoms were in the chest and the arthritic symptoms did not commence until seven years before he came to us for treatment. Since that time—and this I think is an important fact that has come out of Dr Clagett's contribution, on which I congratulate him—we have had 4 other cases on which we have been able to register clinically the diagnosis prior to operation, based on the points Dr Clagett has made.

The interesting thing to me is this question of the benignancy or malignancy of these tumors. In one of my patients who had rheumatoid symptoms with swollen joints for many years I did a local removal of the tumor but I have a vague suspicion that I left part of it behind. Exactly the same thing happened in this patient that Dr Clagett mentioned, the rheumatoid symptoms disappeared completely after removal of the tumor. In a matter of a few months after removal of the tumor this patient went back to her doctor and said,

"Doctor, I am getting my symptoms back, my joints are beginning to get swollen, I cannot get my rings on." The doctor sent her back to me. On x-ray examination it was found that she had a recurrence. When I re-operated on her I had to do a pneumonectomy because the tumor was infiltrating the lung tissue. As a result of the pneumonectomy the joint symptoms have completely disappeared. She is a dear fat old lady, she is short of breath but aside from that she is in pretty good fettle.

The point Dr Clagett has raised with regard to the cause of these arthritic symptoms I think is important. I am surprised at the relative infrequency of these arthritic symptoms in his cases of carcinoma of the lung. Certainly in our country they are more common in carcinoma of the lung than Dr Clagett has mentioned, 4 cases in 200, I think give the figure. But the important thing to me seems to be that you can get

identically the same symptoms and the new subperiosteal bone formation occurring with a series of different conditions. We get them with carcinoma of the lung undoubtedly, I have seen them with a mediastinal sarcoma, with infective conditions in the lung or pleura. I would like to postulate that it is probably not the secretion from the tumor which produces the arthritic symptoms but that the abnormal secretion from the tumor merely acts as a trigger mechanism which starts off something that produces symptoms like a rheumatoid condition. In other words, there is some sort of link in the chain farther back which can be activated by the three or four conditions which may produce the same type of symptoms.

Before I sit down I would like to reinforce what your worthy secretary has said about our own Society. He apologized to me because he called it an association. I myself am never quite sure what we call ourselves. I would also like to say how pleased I am to be here, it is a red letter day for which I have waited twelve years. Further, I would like to say that any of your members who happen to be travelling in our country at the time our Society meets will meet with a very warm welcome. If you are dreaming of coming to England—God knows why you should because we have no meat there—round about November, I suggest that you make your first port of call our Society meeting and you will be able to meet all those of us who are working in this field, and will be able satisfactorily to arrange your schedule for the rest of your trip.

RESECTION OF THE TRACHEA FOR CARCINOMA

REPORT OF TWO CASES

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TWO cases of primary carcinoma of the intrathoracic trachea, in which treatment consisted in complete circumferential resection of the trachea and reconstruction with a stainless steel tube, are presented. Previously reported cases of tracheal resection for cancer are briefly summarized. References examined during an exhaustive search of the recent literature on malignant lesions involving the trachea and on surgical reconstruction of the trachea are listed chronologically.

Primary tumors of the trachea are rare. Ellman and Whittaker³⁵ reviewed the 507 cases of this nature that had been reported up to 1945. Clagett and his co-workers³² reported twenty-seven cases from the Mayo Clinic. The three most common primary tumors of the trachea are squamous cell carcinoma, adenocarcinoma, and cylindroma. The prognosis in cases of cylindroma seems to be better than in other malignant tracheal lesions. In 70 per cent of cases the tumor is situated in the middle or lower third of the trachea.

Treatment for primary malignant lesions of the trachea may be divided, categorically, as irradiation with x-ray, implantation of radon, endoscopic removal of the lesion with forceps or cautery, and surgical removal. The latter comprises three types of procedure: tracheotomy with curettement and cauterization of the lesion, partial circumferential or 'window' resection of the trachea, complete circumferential or divisional resection of the trachea.

Of the therapeutic methods listed, complete circumferential or divisional resection would seem more nearly to achieve the ideal, that is, complete extirpation of the malignant tissue and an adequate margin of normal tissue. The hazards which may be encountered during this procedure, as well as the difficulties of technique may lead, however, to temporization with a method that, while facilitating repair, may constitute inadequate removal of the lesion.

Several cases have been reported of partial or 'window' resection of the cervical trachea after a tracheotomy had been done because of cancer. A number of authors have described reconstructive procedures following resection or trauma. In 1898 Bruns² reported a case in which ten rings of the cervical trachea were resected for a malignant lesion of the thyroid. A tracheal cannula of material not specified was used as a prosthesis. His patient was well for five years following the operation, then a stricture developed and he died. In 1927 Churchill (cited by Rob and Bateman)³ rebuilt the cervical trachea with

¹ Read at the Thirty-Ninth Annual Meeting of the American Association for Thoracic Surgery, Dallas, Texas, May 1947.

a tube of skin following complete traumatic rupture with retraction of the ends of the trachea. Rob and Bateman³⁰ used tantalum gauze and fascia lata for reconstruction of the cervical trachea following resection for a malignant lesion of the thyroid. Crafoord²⁹ rebuilt the cervical trachea with a cutaneous flap containing an implant of costal cartilage.

Resection of the intrathoracic trachea for primary malignant lesions of the trachea has been reported by six authors. In 1945 Belsey³⁸ rebuilt the intrathoracic trachea with wire reinforced fascia following partial circumferential resection for a primary tracheal lesion, the patient died two years later from metastasis. Clagett's³² patient had a tracheal cylindroma. Following complete circumferential resection of the intrathoracic trachea in 1946, reconstruction was performed, using a Polythene tube as a prosthesis, however, the patient died on the sixth postoperative day. Jarvis⁴⁷ completely removed 4 cm of the intrathoracic trachea for a cylindroma and reconstructed the trachea with a stainless steel tube, his patient died of metastasis 33 months later. Belsey,³⁸ in 1949, did a partial circumferential resection of the intrathoracic trachea for cylindroma and repaired the defect with wire reinforced fascia, this operation had been preceded by right-sided pneumonectomy six years before. The patient was alive at the time the case was reported, but the exact period of survival was not indicated. In 1949, Keigin⁴⁸ removed a segment of the intrathoracic trachea for squamous cell carcinoma just above the carina and repaired the trachea with a graft of pericardium. Four months later, because of recurrence of the tumor, a pneumonectomy on the right side was done and an autogenous bronchial graft was applied to the trachea. The patient died, however, on the third postoperative day. In 1950, Cahan⁴⁵ resected the intrathoracic trachea for adenoid cystic carcinoma. A graft of tantalum gauze and fascia lata was used as a prosthesis. Ravitch⁴⁶ mentioned two cases of carcinoma involving the trachea. Paulson used a wire reinforced fascial graft for reconstruction. Ravitch reports tantalum mesh and a dermal graft for reconstruction.

Three of these intrathoracic resections were complete circumferential or divisional resections (Clagett,³² Jarvis,⁴⁷ Ravitch⁴⁶). Only one report (Jarvis⁴⁷) indicated a successful initial result.

In the cases of primary carcinoma of the intrathoracic trachea presented here, a combined cervical and sternum splitting incision was employed, with a right-sided thoracotomy through the second anterior interspace. A complete circumferential resection of the trachea was done and a stainless steel tube was employed for reconstruction of the air passage. In both cases the results have been encouraging. The patients are alive and well sixteen and six months, respectively, following extirpation of their malignant tracheal lesions. Neither has had recurrence of symptoms, evidence of reactivation of the malignant process has not appeared.

CASE REPORTS

CASE 1.—In January 1951 T. P. M., a 58 year old white man, was seen with the complaints of wheezing, coughing, hemoptysis, considerable difficulty in breathing, and a loss of 25 pounds. He could walk only about 20 yards.



Fig 1—Case 1. Preoperative film showing bilateral emphysema due to the partially obstructive tracheal lesion.



Fig 2—Case 1. Lateral view showing tumor mass.



Fig 3—Case 1 Fourth postoperative day showing the subcutaneous emphysema and position of the three inch tube.



Fig 4—Case 1 Lateral view of Fig 3



Fig. 2—Case 1 The tube is shown displaced downward with angulation to the right. The wire sutures can be seen above the tube.



On examining the patient, his recent loss in weight was obvious. A marked stridor was noted, but evidence of metastasis was not found. A roentgenogram of the chest demonstrated emphysema of both lungs. On bronchoscopy a tumor was seen just below the cricoid cartilage which almost completely filled the lumen. Biopsy of the tumor was reported as carcinoma.



Fig 7—Case 1 New four inch tube anchored in place.



Fig 8—Case 1 Lateral view of new tube

On Jan. 15, 1951, the trachea was exposed by a midline cervical incision. After the trachea was opened, the lesion was found to extend almost to the carina. A sternal splitting and right thoracotomy incision was made into the second interspace anteriorly. The trachea was resected from the cricoid cartilage to a point 2.5 cm. above the carina, the entire circumference of the trachea being removed.

Reconstruction of the air passage was effected by inserting a stainless steel tube. Intravenous anesthesia was used, and adequate oxygenation was provided through a long, sterile, endotracheal catheter inserted through the wound and held snugly in the lower part of the trachea by umbilical tape. A stainless steel tube 7.5 cm long, which extended 0.5 cm into each tracheal segment, was sutured in place with multiple wire sutures. Since a tracheostomy could not be done because of the extent of the resection, an indwelling endotracheal catheter, passed through the nose into the trachea, was employed for three days to control the mucus and effect free passage of air. It served also to prevent the patient's closing his vocal cords when coughing thus preventing, in turn subcutaneous emphysema. On the third postoperative day, however, subcutaneous emphysema did appear which was thought to be caused by obstruction of the endotracheal catheter. This proved to be the case. The lower end of the catheter was almost completely obstructed by blood and mucus. It was not found necessary to reinsert the catheter. The remainder of the patient's hospitalization was without complications, and he was discharged on the tenth postoperative day.

After an uneventful convalescence, with complete relief from his symptoms, the patient returned to full time work.

Nine months later, in September, 1951, while painting a ceiling, the patient inhaled some dust which caused a severe attack of coughing. For the next few days he had an irritating cough, he was also having difficulty on inspiration. Roentgenologic examination revealed that the steel tube had slipped downward about 3 cm., both lungs were adequately aerated. Bronchoscopy confirmed that the tube had slipped, the exposed portion of the reformed trachea was mobile and was filled with polypoid granulation tissue. Many polypoid masses were removed, following which the patient's symptoms were somewhat relieved. On the two succeeding days additional polypoid masses were removed bronchoscopically. Microscopic studies of the tissues revealed no recurrence of the malignant growth. Further symptomatic relief had been effected by the repeated bronchoscopic therapy, however, about ten days later increasingly severe stridor appeared. Bronchoscopy at this time revealed a firm annular stenosis with a lumen of only 3 to 4 mm. On Sept. 21, 1951 he was operated upon. The tube was found to have slipped. The stenotic area was curetted and the tube resutured into place with No. 0 cable wire. The patient had a complete and uneventful recovery and resumed full time work in an airplane factory. All of his previous symptoms disappeared.

On April 7, 1952 he contracted a severe upper respiratory infection which was accompanied by a great deal of coughing. Following this, stridor developed. Roentgenologic examination indicated the tube had again slipped and that stenosis had developed at the same site as before. On April 15 a new tube was inserted and anchored in place with No. 28 wire sutures. He immediately lost all his symptoms. He was able to leave the hospital in five days. Twelve months after this time again were free of evidence of recurrence of the carcinoma. The entire air passage still was mobile and was lined with polypoid masses of granulation tissue with exception of the upper 3 cm. which constituted a heavy rigid fibrous wall that was the site of the stenosis.

Sixteen months now have elapsed since removal of the malignant lesion. There is not, at present, any evidence of recurrence.

The wire sutures used in the previous operations were in each instance found to be lacking. In retrospect I feel that the suture material was not strong enough to anchor the tube. We hope that the No. 28 wire will be adequate. At no time has there been evidence of cancer at either end of the steel tube where it is in contact with the tracheal mucosa.

CASE 2.—On Oct. 30, 1951, W. W., a 53-year-old white man was seen with the complaint of a dry, tickle over shooting of four months duration. For the last two weeks he had had frequent respiratory embarrassment. He was being treated for asthma.



Fig. 9—Case 2 Postoperative film showing two inch tube in place.



Fig. 10—Case 2 Lateral view of Fig. 9

The patient appeared well nourished. He had a marked stridor. Roentgenograms of the chest were essentially normal. Bronchoscopic examination revealed a tumor in the middle third of the trachea which had the typical appearance of a bronchial adenoma. It almost completely obstructed the lumen except for a narrow passage along the right wall. Biopsy was reported as benign tracheal adenoma. However, and in the face of opposition from the entire hospital tumor board it was decided to perform a complete circumferential divisional resection of the trachea.

(On Nov. 1, 1951, a segment of the thoracic trachea 3.5 cm. long was removed and reconstruction of the trachea was effected by inserting a stainless steel tube 5 cm. in length in a manner similar to that employed in Case 1. Histologic examination of the tissues removed revealed the lesion to be an adenocarcinoma. As in Case 1, an indwelling endotracheal

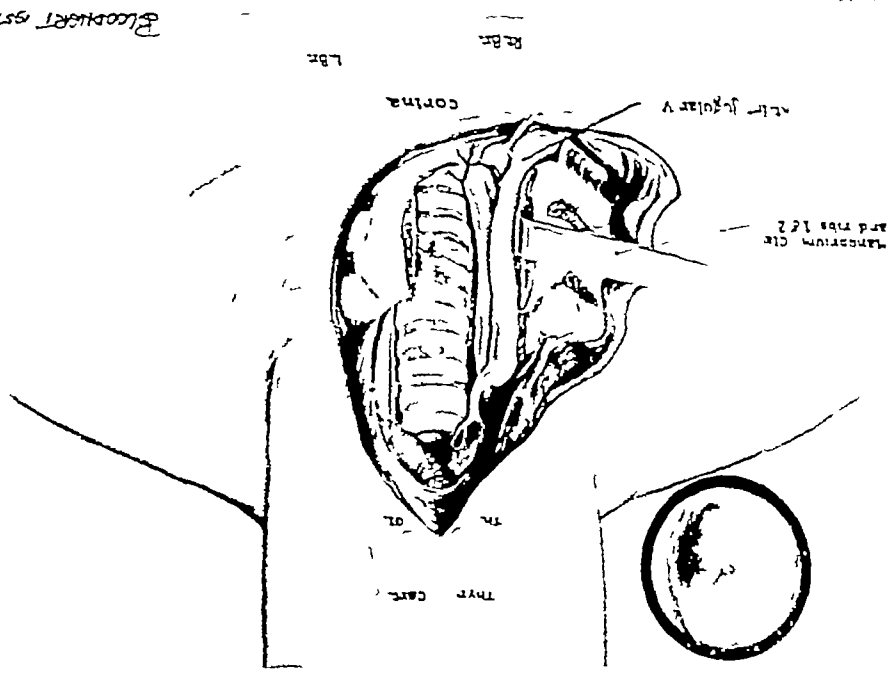


Fig. 11-1. A view of the trachea through a cervical sternal splitting and right thoracotomy incision. The incision is a bronchoscopic view of the lesion.

At the time of the operation the most extensive removal of tumor was postoperatively. This was done by removing the fourth to sixth ribs which were found to be obstructed by a laminated clot of fibrin. The patient's postoperative course was uneventful. The trachea and the left lung were found to have remained in place and there was no evidence of emphysema. The patient was able to have the operation. On bronchoscopic examination in April 1952 a combined cervical sternal splitting incision with a right sided thoracotomy through the second intercostal space gave adequate exposure for resection of the trachea.

Postoperatively an indwelling endotracheal catheter introduced through the trachea permitted adequate control of mucus and prevented closure of the

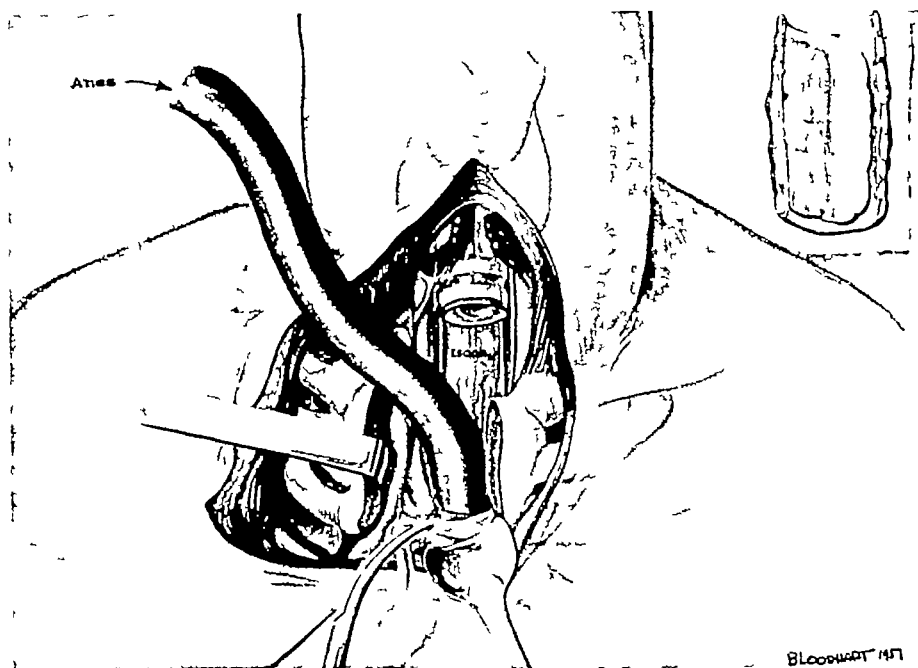


Fig. 12—Trachea resected with lesion shown in the insertion. Long anesthesia tube shown held in lower end of the trachea.

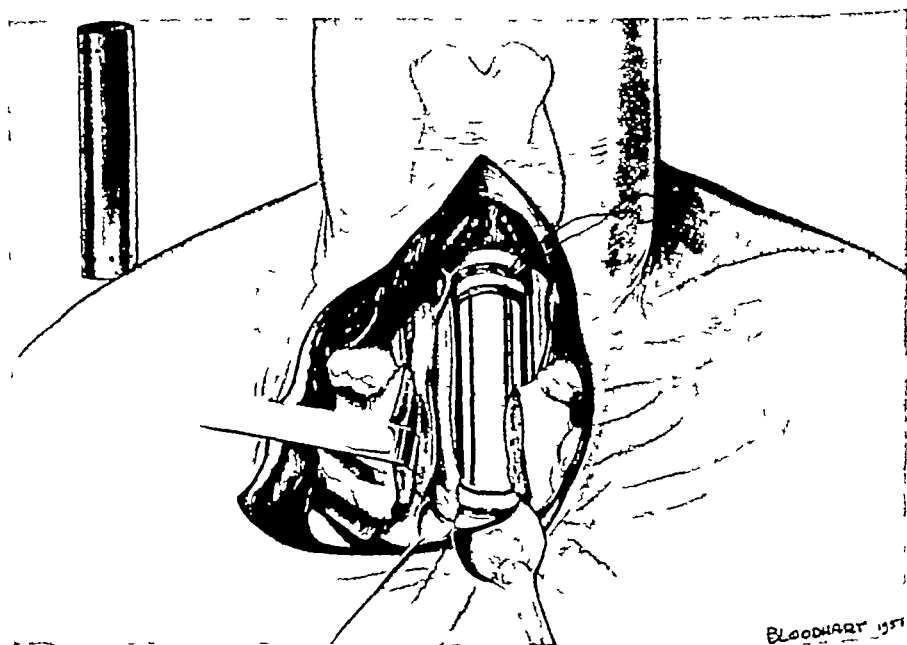


Fig. 13—Three-inch stainless steel tube in place.

vocal cords, thus minimizing subcutaneous emphysema, the catheter was well tolerated by both patients. It had to be removed three and four days, respectively, following operation because of obstruction by blood-stained mucus. In neither instance was reinsertion of the endotracheal catheter required.

Because mass ligatures were not used in either case, the closure of the trachea was not airtight, however, subcutaneous emphysema did not occur until the third day.

Intravenous anesthesia was used. Adequate oxygenation was provided by the use of a supplementary, sterile, long endotracheal tube inserted through the operative wound and held snugly in the lower part of the trachea by umbilical tape.

Postoperative infection was not encountered. Penicillin, 100,000 units every three hours, was employed during the postoperative period.

A stainless steel tube was anchored with mattress wire sutures through holes in the tube and then through the trachea. The tube extended 1 cm into each segment of the trachea. No. 28 wire proved to be the best choice of suture material.

SUMMARY

1. Two cases of combined cervical and intrathoracic resection of the trachea are presented. Both patients are alive and well sixteen and six months, respectively, following extirpation of the malignant tracheal lesion.

2. Complete circumferential or divisional resection more nearly achieves the ideal treatment for primary neoplasm of the trachea than does any other method of therapy.

3. Reconstruction of the trachea with a stainless steel tube was adequate.

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DISCUSSION

DR O THERON CLAGETT, Rochester, Minn.—My colleagues and I have been interested in this problem of tracheal tumors for some time, having begun the development of techniques for the reconstruction and repair of the trachea in 1946. To date we have performed operations on four patients with tracheal tumors. In our total experience with tracheal tumors the most common type is squamous cell carcinoma. Cylindroma is the second most common, adenocarcinoma is third, and hemangioendothelioma is fourth. We do not believe that it is technically possible to perform a really radical operation, in terms of cancer

urgency, for tumors of the trachea. The best that can be accomplished is a local excision of the growth. For this reason we believe that resection of cylindromas of the trachea offers our best surgical opportunity since they are slow growing tumors which metastasize late.

(Of the four patients upon whom I have operated, three had cylindromas, one was squamous-cell carcinoma. All of these tumors involved the right lateral wall of the trachea. In our first case it was fortunate since this region is most accessible to surgical attack. The trachea was joined to the left main bronchus with a long Polythene tube. This patient died of pneumonia on the sixth postoperative day due to obstruction of the left upper lobe bronchus by the tube. Our second patient had a small tumor. A window resection of the right lateral wall of the lower trachea was carried out and the defect was repaired with a fascial patch reinforced and contourered with steel wire. This patient is still well two and one-half years after operation. Our third patient had a squamous-cell carcinoma involving the right lateral wall of the middle third of the trachea. Regional lymph nodes were involved. A large window of trachea was resected. This defect was too large for repair with a fascial patch so a Polythene tube was inserted. This patient died of recurrent and metastatic carcinoma about two years later. However, the Polythene tube provided adequate airway throughout most of this period. Our fourth patient had a large cylindroma involving the right lower trachea and part of the right main bronchus. The tumor was resected and the defect repaired with a wire reinforced fascial patch. It was necessary to remove the wire from the trachea a few months after operation by means of bronchoscopy, due to over granulations at the site of repair and stenosis.

I do not believe that a wire reinforced fascial patch is a satisfactory method of repair for large tracheal defects. However, it has worked well clinically and experimentally for all defects. Our experimental results with polythene tubes has been very satisfactory. (The clinical results have not been discouraging). However, we must continue our search for better methods for repair and reconstruction of the trachea. Tracheal tumors are rare, but I think who live them deserve our best efforts to relieve them of their progressive coughing paroxysms. A technique I point that should be mentioned as to emphasize the importance of performing careful tracheotomy at the time of operation in every case. This procedure will undoubtedly be valuable to the extent of the patient during the early post-operative course.)

PAPER READ BY RAY CLARKE—I certainly enjoyed Dr Cotton's excellent paper. I had no even interest in this problem for the past several years and have operated upon seven patients with various types of tracheal lesions. Four patients had extensions of bronchogenic carcinomas into the lower trachea; from lesions originating in the right lung which I treated or clipped extensive excisions of the lower trachea and carina. The other three I treated by removal of partial interest in tracheal surgery and I would like to state that the operative procedures employed because of the incidence of complications associated with the use of prostheses the surgical reconstruction of the tracheobronchial tree in such cases was accomplished by the adequate mobilization of the proximal and distal segments.

DR. FREDERICK BAX (London) said—I certainly enjoyed Dr Cotton's excellent paper and I am interested in this problem for the past several years and have operated upon seven patients with various types of tracheal lesions. Four patients had extensions of bronchiectasis into the lower trachea from lesions originating in the right lung, although one had widely or elliptical extensions of the lower trachea and carina. The other three patients are of particular interest in reference to tracheal surgery and I would like to start with the use of prostheses, the surgical reconstruction of the tracheobronchial tree in which was accomplished by the adequate mobilization of the proximal and distal segments and primary anastomosis.

The first patient to be discussed had a large bronchoma of the upper trachea. The trachea, including the upper and lower tracheal rings and posterior one-half of the cricoid cartilage, was removed. The distal trachea and upper stem bronchi were mobilized fully for all was for a primary anastomosis. A temporary tracheostomy was performed and the anastomosis. The thing was excellent. There has been no evidence of stenosis or stricture at the anastomosis. He also followed up

was instituted for four days postoperatively. The convalescence was uneventful. The patient is alive one year postoperatively without evidence of recurrence at the primary site but with evidence of mediastinal glandular metastasis.

The third patient was operated on about a month ago. He had a carcinoma of the right lung which had extended into the lower trachea and left main stem bronchus. A right pneumonectomy was performed including about one inch of the lower trachea and about two thirds of the left main stem bronchus. Then by mobilizing the trachea above and the entire hilar region of the left lung, sufficient mobilization was obtained to approximate the ends. As is seen in the slide of the surgical specimen the disproportion between the larger tracheal lumen and the smaller size of the bronchus was overcome by invaginating the distal bronchus into the trachea and then performing an anastomosis by individual suture technique. As is noted on the screen the anastomosis was just above the left main stem bronchus. This patient died six days postoperatively from an acute myocardial infarction. Healing of the anastomosis appeared excellent for this stage.

DR OSLER ABBOTT, Atlanta—There are two matters felt to be worthy of discussion. One is that I do not think discussion of this problem will in any way be complete unless we pay due credit to Rollin Daniel for the work he presented to this Association on the experimental aspect. Two, I thought it might be of some interest to let you know of the follow up on the six patients on whom we did resection of the lower trachea and carina and reported to the Association, by title, three years ago. It only adds emphasis to what has been said, and for that reason I bring it up.

Five of the cases of carcinoma with extension were brought to light because of the enthusiasm of the young surgeon. The palliation was not justified. Prostheses with glass tubes were used in two patients, one of whom developed discomfort. The tube was removed, apparently ill advisedly, after nine months, and was followed by stenosis and death, no post mortem was done. The sixth patient had a cylindroma, primarily of the carina. This patient had a right pneumonectomy, and had a reconstruction of the carina by the early method that we described for repair of the trachea and left main stem bronchus. That repair gives a very difficult postoperative course and for that reason is to be condemned. Other than that, the ultimate result was excellent. This patient with a cylindroma lived for three years and nine months and died from abdominal metastasis after an x-ray diagnosis had revealed carcinoma of the stomach. So, for this reason, I feel I could emphasize Dr Clagett's point to use this as much as possible for relatively benign lesions.

DR J F MAC MANUS, Buffalo, N. Y.—I would like to subscribe to Dr Cotton's principles and those of Dr Kay in the handling of primary lesions of the trachea. I believe we should treat them as we do other tubular organs, with segmental resection and whatever nodal resections seem appropriate. In view of the fact that the esophageal chain, the tracheal chain and the hilar bronchial chain are so intimately associated, it is not very likely that we will do adequate cancer surgery, but I do believe that segmental resection with primary anastomosis should be our objective.

I would like to add one case to Dr Kay's cases that he has presented, of a patient who had a primary lesion of the trachea with a segmental circumferential excision of the lesion and a primary anastomosis. This was an intrathoracic lesion measuring approximately 3.2 centimeters in size.

(Slide.) This is the lipiodol study of the patient before surgery, a 59 year old man with hemoptysis as the only symptom. The lipiodol runs down the anterior wall of the trachea and you can see it outlining the tumor quite satisfactorily. We did this because we wanted to be certain of the exact location of the lesion, feeling that since the pathologist reported a very mature squamous cell lesion, we might have difficulty in finding it with the intratracheal tube in the trachea if we did not know exactly where it was beforehand.

(Slide.) This shows the resected lesion. The segment comprised three cartilages anteriorly and six posteriorly. As we all know the cartilage bifurcates shortly after passing lateralward. That amounts to a segment of trachea approximately 3.2 centimeters in width.

(Slide) This demonstrates the lesion to be adequately resected, at least locally, by turning it inside out and allowing us to view the area of local attachment

(Slide) After studying the sections our pathologist changed his diagnosis from a very mature squamous cell carcinoma to a cylindroma, but I believe, as many of you do, that the cylindroma is fundamentally a malignant tumor

(Slide) This is the trachea after resection. One can see the outline of the tracheal wall. By direct endoscopic visualization the suture line was entirely healed in seventeen days with no loss of lumen.

DR OTTO C BRANTIGAN, Baltimore—Dr Cotton in his very concise manner has given us as usual an excellent presentation, and I wish to congratulate him on the good results he has obtained. I arise merely to say a word about our experience in work on the trachea. Experimentally, Dr Penton conceived the idea of a viable, living pedicle made from the chest wall. The pedicle includes pleura, periosteum, intercostal artery, nerve, vein and intercostal muscles. We could remove in the dog the complete circumference of the trachea or any part of it and repair the defect with the viable pedicle and have uniform recovery of the animal. We have not had a patient with carcinoma of the trachea. However, in dealing with an individual with carcinoma of the esophagus where we got into a situation that had to be finished, we had the opportunity of applying this technique when an extensive excision of the trachea had to be repaired.

After excision of the carcinoma from the trachea there was a defect of more than half the posterior circumference of the trachea and both major bronchi, and extending a distance of about one inch on both major bronchi and about two to two and one-half inches of the lower end of the trachea. The viable pedicle flap of the chest wall was used to repair this defect. No support was used in the flap. However, a soft rubber tube was placed in each bronchus and brought out by way of the trachea through a tracheotomy wound in the neck. These tubes were removed within six days. The patient made an uneventful recovery. Unfortunately, he died of generalized metastasis about four months after operation.

DR. COTTON—(Closing.) In closing I would like to show a specimen to demonstrate how clean the tube remains.

(Slide) This stainless steel tube was inserted for an aneurysm which was causing tracheal obstruction. The patient died three months after insertion, but I show this to demonstrate how clean the tube remained even though it was in the trachea for three months.

May I thank the discussors for their remarks.

THE SURGICAL TREATMENT OF CARCINOMA OF THE HYPOPHARYNX, LARYNX, AND UPPER ESOPHAGUS

ORVILLE F. GRIMES, M.D., AND H. BRODIE STEPHENS, M.D.
SAN FRANCISCO, CALIF.

THE over all decrease in the mortality rates for surgery of the esophagus and hypopharynx has encouraged an ever broadening horizon with respect to the aggressive surgical attack on such lesions. The rare operation of ten years ago has now become an almost commonplace procedure. Credit for this belongs not only to those who have perfected the technical details of the operative procedures but also to improvements in anesthesia and to the contributions of physiologists who have solved so many of the problems of the pulmonary and cardiovascular systems. Chemotherapy and the antibiotic agents have added their increment of improvement in combating and preventing infections, and adequate whole blood replacement therapy during surgery has permitted the confident performance of a deliberate and unhurried operative procedure.

Cancer of the hypopharynx, upper esophagus, and larynx is more closely related to the intra-oral group of carcinomas than to the gastrointestinal malignancies of glandular origin. The resemblance is evident in that epidermoid carcinoma is the common histologic type, the age and sex distribution are similar, and there appears to be a common etiologic relationship to chronic irritants of various sorts. Contrary to true esophageal cancer which often metastasizes early and widely, carcinoma which arises in the larynx and spills over into the hypopharynx or that which arises in the hypopharynx and secondarily involves the larynx or the upper esophagus often tends to remain localized, and its metastases frequently are limited to the cervical lymphatic chains for a considerable period before ultimate more rapid distant dissemination occurs.

During the past three years (from March, 1949, through March, 1952) seventeen patients with advanced malignant lesions involving the hypopharynx, larynx, and upper esophagus have been treated surgically at the University of California Hospital. The procedure popularized by Wookey¹ of Toronto has been applied and in the majority of the patients treated has been extended to include a unilateral radical neck dissection. The operation is designed to provide a wide en bloc excision of all malignant tissue.

A rectangular flap of skin and subcutaneous tissue is raised on the anterolateral surface of the neck with its hinge on the side corresponding to the location of the lesion within the hypopharynx. If definitely enlarged cervical lymph nodes are present unilaterally, a standard radical neck dissection is accomplished before the site of the primary lesion is attacked.

After completion of the neck dissection, the isthmus of the thyroid gland is divided along with the sternal attachments of the infrahyoid musculature.

¹From the Department of Surgery, Division of Thoracic Surgery, University of California School of Medicine.

Read before the Thirty-second Annual Meeting of The American Association for Thoracic Surgery, Dallas, Texas, May 8-10, 1952.

The trachea is preferably divided at the cricoid cartilage, but may be transected at a much lower level depending upon the downward extent of the tumor. Such extension usually is manifested externally by a mass intimately adherent and roughly parallel to the larynx and trachea. Anesthesia is thereafter administered through a right-angled, inflatable euffed rubber tube placed directly into the open end of the trachea which is exteriorized in the region of the suprasternal notch. The esophagus is transected at a similar level. All or part of the contralateral thyroid lobe is preserved mainly to insure adequate para-thyroid function.

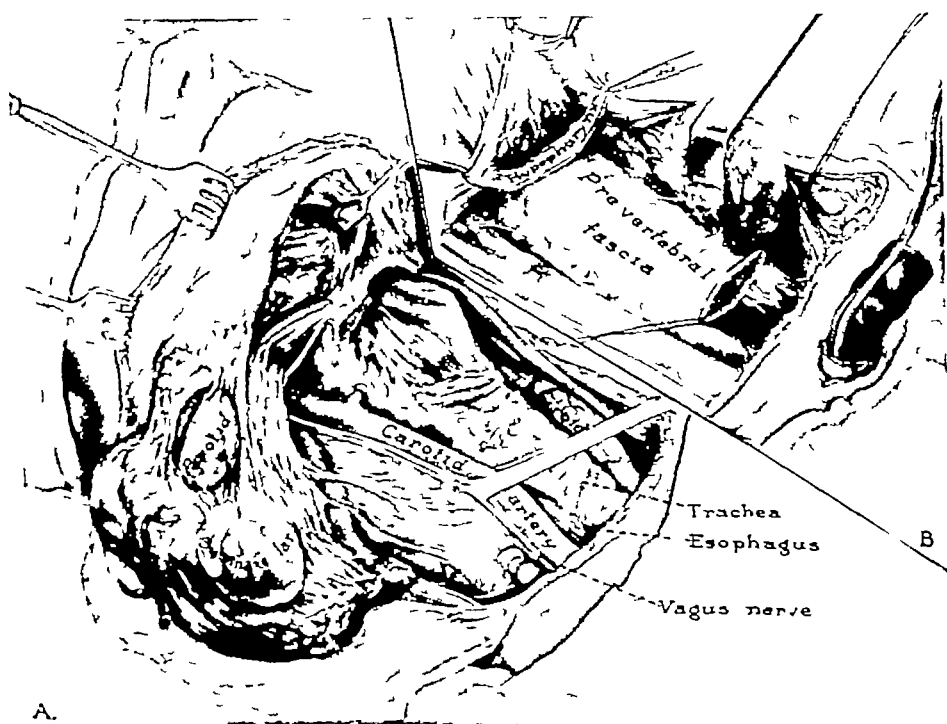


Fig. 1—1. The skin flap has been reflected toward the right side along with the contents of the radical neck dissection. B. The tracheal segment, upper esophagus, larynx, and hypopharynx have been removed en bloc on the plane of the prevertebral fascia. The trachea has been exteriorized.

An upper incision is carried into the hypopharynx through the thyrohyoid membrane. In most instances the hyoid bone can be preserved. However, partial or total excision of the hyoid is feasible if warranted by the extension of the lesion and surprisingly enough produces little or no difficulty in swallowing.

The plane of dissection once the trachea and esophagus are transected inferiorly is easily established. The entire tracheal segment, larynx, hypopharynx, and upper esophagus are rather easily mobilized and removed on the plane of the prevertebral fascia. The hypopharynx is transected as far superiorly as possible. Almost always it is wise to remove a portion of the base of the tongue (Fig. 1).

Occasionally the carcinomatous lesion may extend upward along the lateral hypopharynx to levels as high as the tonsillar fossa. Regardless of the severity of the proximal involvement, adequate triangular excision of the malignant extension can usually be accomplished (Fig 2). The repair of the resulting hypopharyngeal defect necessarily decreases the caliber of the pharyngeal stoma, but apparently not excessively so.

The first stage of the procedure is completed by partially reconstituting the swallowing mechanism. The flap of skin and subcutaneous tissue is utilized to form a skin-lined swallowing tube. The flap is swung back across the prevertebral fascia and sutured circumferentially to the hypopharynx above and to the transected end of the esophagus below, forming what is termed the "lateral

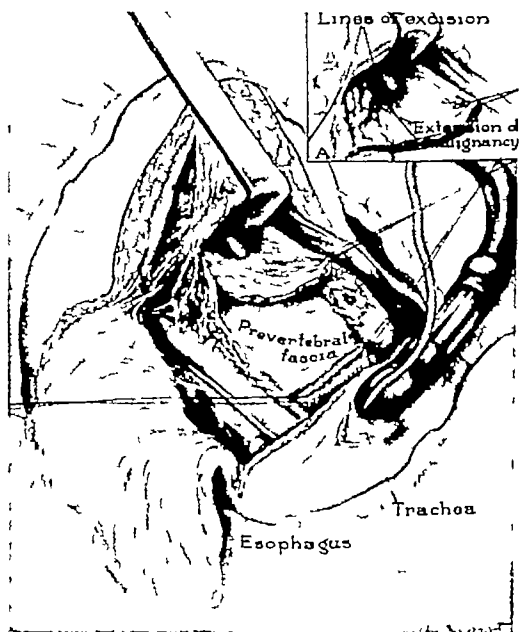


Fig 2—Large segments of the lateral hypopharyngeal wall and the base of the tongue frequently must be excised because of the proximal extent of the lesion.

sinus (Fig 3). A Polyethylene tube is inserted through this area into the stomach for feeding purposes during the interval between the first and second stages of the procedure. The excess margins of the skin flap are sutured to the surrounding skin edges (Fig 4). Frequently skin grafts are necessary to close the raw areas. The lower regions of the wound receive soft rubber drains.

A waiting period of three months is usually necessary before the second stage of the procedure may be safely accomplished. During this interval the blood supply of the partially formed swallowing tube is insured by the development of new vascular channels directly from the tissues deep to the transposed skin flap.

The swallowing mechanism is completed by appropriate vertical and horizontal incisions designed to mobilize enough of the skin flap to close the

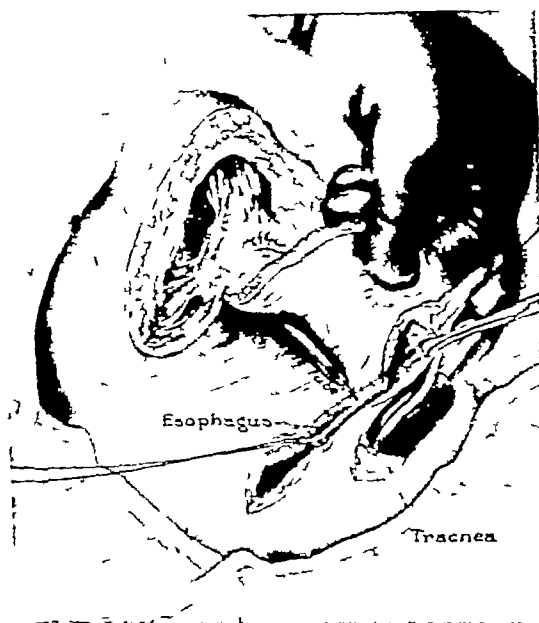


Fig. 3—The circumferential union of the skin flap to the hypopharynx and esophagus forms the lateral sinus.



Fig. 4—After insertion of a polyethylene catheter through the completed skin lined flap, the stomach for esophageal tube, the surrounding skin edges are sutured to the suture line of the skin flap.

lateral sinus In the majority of instances the closure is easily performed, since the circumferential suturing both to the hypopharynx and to the esophagus has been completed at the initial stage of the procedure However, necrosis from an inadequate blood supply may follow the extensive neck dissection necessary to excise large metastatic cervical masses, and a portion of the skin flap may be lost In such instances, the skin-lined swallowing tube may still be completed by a wider mobilization of skin and subcutaneous tissue A double row of nonabsorbable sutures is preferred The raw external surface is covered by a skin graft of intermediate thickness An ideal dressing with localized pressure is provided by fluffed gauze secured with the sutures used to anchor the skin graft

Twelve patients of the seventeen operated upon are alive from one to thirty-six months after surgery (Table I) Nine of these patients are well, and have shown no evidence of recurrence for from one to twenty four months Some have returned to gainful employment Three patients have tiny pin point fistulas which thus far persist in spite of all efforts designed for their closure Fortunately, however, they experience little or no difficulty in swallowing

TABLE I HYPOPHARYNGEAL CARCINOMA ANALYSIS OF SURVIVING CASES

	NAME	SEX	AGE	PRIMARY LESION	TIME SINCE OPERATION (MONTHS)	COURSE	PRESENT STATUS
1	Q Q	M	48	Esophageal carcinoma	36	Lumbar spine metastases 20 months after surgery & with roentgenogram	1 ? Bone metastasis left femur 2 Probable cervical lymph node metastases
2	E R	M	52	Hypopharynx	24		1 No evidence of recurrence 2 At work
3	A W	M	70	Invasive thyroid carcinoma	17		1 No evidence of recurrence 2 Small salivary fistula
4	P R.	M	71	Hypopharynx	13		1 No evidence of recurrence 2 Small salivary fistula
5	W J	M	49	Hypopharynx	12		1 No evidence of recurrence 2 At work
6	W K	M	55	Hypopharynx	11		1 No evidence of recurrence 2 Small salivary fistula
7	D S	M	71	Hypopharynx	11		1 Cervical lymph node metastases
8	I M	M	53	Hypopharynx	4	Hemorrhage 4 months after surgery	1 Recurrence, local
9	R M	M	70	Hypopharynx	5		1 Well
10	H M	M	52	Hypopharynx	2		1 Well
11	F McF	M	49	Hypopharynx	1	One stage procedure	1 Well
12	G L	M	52	Hypopharynx	1	One stage procedure	1 Well

The first patient in this series (Q Q) presented the problem of cervical esophageal carcinoma with direct extension into the larynx. The lowermost level of the lesion in this 48 year old Chinese laundryman was at the level of the clavicle. Because of the length of the involved area, the skin flap was of greater width than usual. Consequently, the trachea was brought out lateral to the usual midline supra-sternal position. Fig 5 demon-

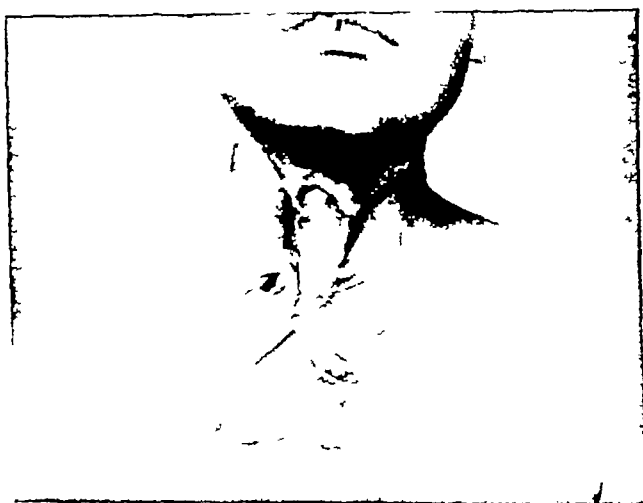


FIG 5—Note the minimal external deformity produced by the extensive removal of malignant tissue

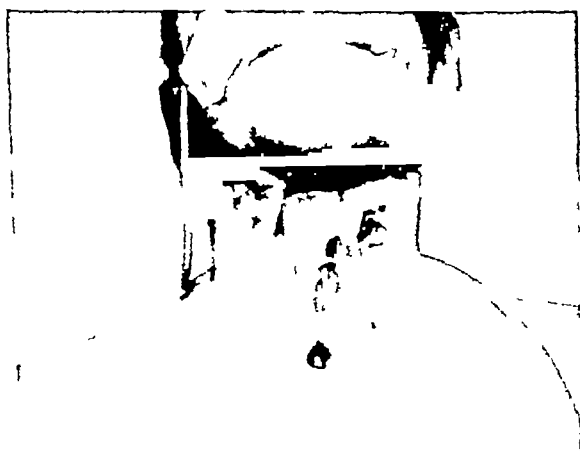


FIG 6—The tracheotomy is visible in the supra-sternal notch. The irregular defects in the anterior wall of the skin-lined swallowing tube healed without impairment of function. The gastric tube traversing the area remained in situ until healing had occurred.

strated the completed second stage of the procedure. The largest raw area produced by the extensive dissection required considerable skin grafting. The skin-lined swallowing tube healed well. The scars have closed since the original operation. Metastases in the larynx and lungs which developed twenty months after surgery are under fair control with nitrofurazone.

A 70 year old man, a teacher of English literature (A W), presented an interesting problem. Three subtotal thyroidectomies for recurrent carcinoma of the left thyroid lobe had been performed since 1936 when the diagnosis was first established. Dysphagia and hoarseness due to invasion of the esophagus and larynx had developed to an alarming degree by November, 1950, when the patient was first seen at the University of California Hospital.



Fig 7—The completed first stage of the procedure. The lateral sinus joins the hypopharynx above with the esophagus below. A right radical neck dissection has been accomplished.



Fig 8—Some degree of narrowing can be expected at the anastomosis of the esophagus with the skin lined tube. All patients in the series have been able to tolerate semisolid to solid foods without difficulty.

Since the invasive thyroid carcinoma failed to take up radioactive iodine in sufficient quantities to be therapeutically effective, extensive surgery was the only alternative. In spite of some necrosis of the skin flap at the second stage of the procedure (Fig 6), the skin lined swallowing tube has healed and functions well. No evidence of recurrence can be demonstrated seventeen months after excisional therapy.

A 71 year old Italian man (P R) with a carcinoma of the hypopharynx and upper esophagus had symptoms for only three months. The malignant lesion extended upward along

the midline of the posterior wall of the hypopharynx to the level of the uvula. The narrowed hypopharyngeal stoma resulting from the excision of the malignant extension (Fig 7) has not interfered with swallowing since the completion of the second stage of the procedure.

A 49 year old white man, a janitor, had an extensive lesion of the right pyriform sinus with unilateral cervical lymph node metastases. Roentgen studies of the skin lined swallowing tube demonstrate the adequate caliber and function of the mechanism (Fig 8). There is no evidence of recurrence, and he has returned to his usual occupation twelve months after the original surgery.

TABLE II HYPOPHARYNGEAL CARCINOMA OPERATIVE DEATHS

	SEX	AGE	PREVIOUS THERAPY	NUMBER OF DAYS	CAUSE
M A	M	54	X ray	54	1 Hemorrhage 2 Empyema
M W	F	68	None	14	1 Hemorrhage 2 Respiratory distress

There were two operative deaths in the series (Table II), one two weeks, the other fifty four days after surgery, both from complications associated with postoperative hemorrhage. One fatality occurred in a 68 year old white woman with an extensive carcinoma of the cervical esophagus with tracheal and laryngeal involvement. Because of the length of the involved area, the trachea and esophagus were transected at a low level below the suprasternal notch. Two episodes of hemorrhage during the first ten days after surgery were controlled with pressure dressings. Bleeding again recurred on the fourteenth postoperative day. The patient apparently became asphyxiated from blood which dropped directly into the low lying trachea in spite of all efforts that were made to prevent such an occurrence.

The other operative death occurred in a 54 year old Italian man who had previously received approximately 12,000 roentgen units to the anterolateral neck regions in an effort to control an extensive carcinoma of the larynx with secondary hypopharyngeal involvement. The presence of metastatic carcinoma in the right deep cervical lymphatic chain necessitated a unilateral neck dissection. Because of the impairment of the blood supply of the skin flap from the previous roentgen therapy and the development of postoperative infection, necrosis of the right common carotid artery occurred. It was necessary to ligate this vessel at its origin from the innominate artery through an anterior thoracic approach. An extensive empyema resulted, causing the patient's death fifty four days after the initial surgery.

TABLE III HYPOPHARYNGEAL CARCINOMA ANALYSIS OF FATAL CASES

	SEX	AGE	PREVIOUS THERAPY	SURVIVAL TIME (MONTHS)	CAUSE OF DEATH
F B	M	57	X ray	12	1 Local recurrence 2 ' Widespread metastases
J N	M	64	0	14	1 Persistence of carcinoma 2 Widespread metastases
A Mc	M	58	0	26	1 Cervical L.N. metastases 2 ' Local recurrence hypopharynx

In addition to the two operative deaths three other patients undergoing surgery for extensive lesions in and about the hypopharynx have died during the period of study. The survival period in the three patients varied from twelve to twenty six months (Table III). In the instance of J N the bilateral extent of the cervical lymph node metastases was not fully apparent at the time of the first stage operation. Because of the persistence of carcinoma in the anterior neck regions and the development of generalized metastatic disease the patient died fourteen months after surgery without ever having the swallowing tube placed. In retrospect surgery should not have been attempted. Two other patients died twenty six months respectively after surgery.

Cervical lymph node metastases were present in fourteen of the seventeen patients in the series. In one instance, already mentioned, bilateral metastases were present, although this fact was not apparent in the original evaluation of the patient. In one case, in spite of the presence of unilateral lymph node metastases, a radical neck dissection was postponed, since the patient's general inventory suggested that the procedure might best be accomplished in stages. After completion of the skin-lined swallowing tube, a radical neck dissection was attempted, but at this late date the metastatic nodes were densely adherent to the right common carotid artery at its bifurcation and could not be removed. The wiser choice would have been the combined procedure at the original operation regardless of the greater risk.

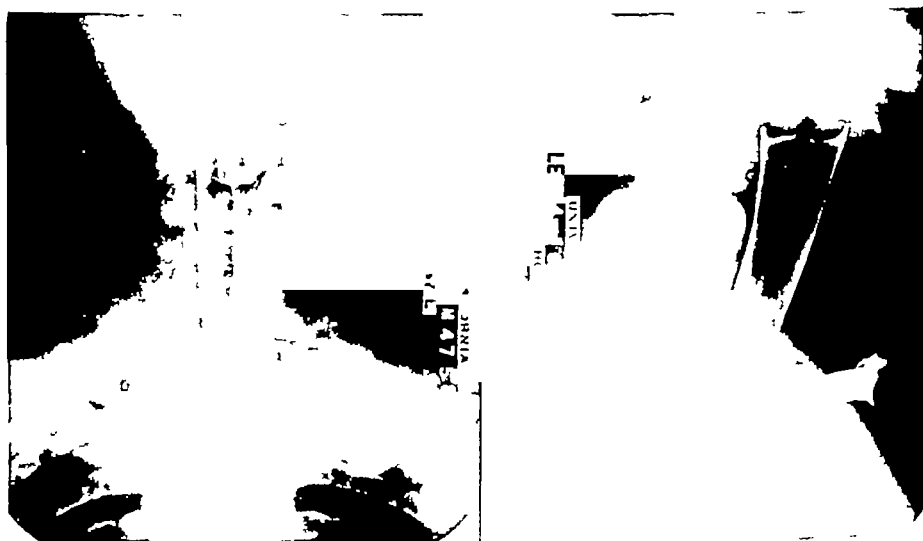


FIG. 9.—The tantalum mesh is shaped to approximate the diameters of the hypopharynx and esophagus. The adequate caliber of the tube is evident.

Recently, the use of splints of various sorts has been proposed to bridge the defect between the hypopharynx and esophagus. We have used prostheses in two patients.⁹ A skin graft of intermediate thickness wrapped about a tantalum gauze mesh splint as suggested by Edgerton was used in both instances. The first patient developed a small salivary fistula that healed within five weeks after surgery. He is now able to tolerate soft foods without difficulty (Fig. 9). The second patient's recovery has been complicated by a rather large fistula at the level of the esophageal anastomosis that persists one month after surgery.

CONCLUSION

With respect to the far advanced malignant lesions for which this extensive operation is performed the results are not particularly discouraging. It is

⁹Dr. M. Galante, Chief Resident in Surgery, performed these two operations.

reasonable to assume that if the principle of wide en bloc excision is applied to earlier more localized lesions the results of surgical treatment will be significantly improved.

As surgery of this magnitude develops to an even greater security, and better results can be expected with increasing frequency, the wider application of extensive surgery for earlier lesions in and about the hypopharynx must be accepted.

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DISCUSSION

DR MARK M. RAVITCH, Baltimore—There are a couple of points of interest in connection with Dr. Grimes' paper and his description of the Wokev procedure. It may be of interest to the members of the Association that in 1891 Von Hacker performed a Wokev procedure quite successfully on dogs and advised its performance on human beings, and, in 1905 reporting in the *Archiv für Klinische Chirurgie* Vol. 57, he performed a full fledged Wokev operation with a square flap laid down as so well described today. That patient was alive and well sixteen months later. Dr. Grimes mentioned Dr. Edgerton's cases, he has now had a considerable experience with replacement of the pharynx and upper esophagus by the tantalum mesh tube which you saw pictured, which is the method of course of Mr. Eric Thomas' compatriots Robb and Biteman, which has been employed for both the esophagus and the trachea. As Dr. Grimes indicates when that method is successful, the patients are very rapidly rehabilitated.

DR H. B. PRODIE STEPHENS, San Francisco—Dr. Grimes is to be congratulated upon his excellent presentation of our results in the application of the Wokev operation in the management of carcinoma involving the cervical segment of the esophagus. Dr. Grimes has demonstrated the utilization of unilateral radical neck dissection in combination with the Wokev procedure.

We all admit that carcinoma in this area presents many problems with regard to surgical treatment and, in addition in our experience the larynx usually has to be sacrificed. Local recurrence rate is high and the result is a low salvage rate. On the other hand, we believe we are obliged to offer these unfortunate victims some ray of hope and some relief, if possible. Metastatic cervical lymph node deposits if unilateral have not been considered a contraindication to operation.

The recent procedure described by Edgerton, namely a one stage reconstruction of the cervical esophagus in which the cervical esophagus is replaced by a split thickness skin graft rolled over a stainless steel wire mesh fashioned into the shape of the removed segment of the esophagus may become a distinct advance over the two stage operation presented to us by Dr. Grimes.

We have had occasion to operate upon two patients employing the technique described by Edgerton. It was necessary to remove the wire mesh three months following insertion of the patient because of infection and low grade inflammatory reaction in the neck. The patient without vibrating the wire prosthesis satisfactorily for a period of two and one-half months. Whether or not a stricture will develop following removal of the wire stent will yet be determined by future observation.

In closing I should like to encourage the members of our Association particularly the younger ones to continue their efforts in the development and improvement of the treatment of carcinoma of the cervical portion of the esophagus.

CARCINOMA OF THE ESOPHAGUS

A CONSIDERATION OF CURATIVE AND PALLIATIVE PROCEDURES

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AND THOMAS N P JOHNS, M D (BY INVITATION)
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THE gloomy predictions of a few decades ago that esophageal cancer could not be expected to yield to surgical attack have proved short sighted and unsound. Such predictions are epitomized in the statement, "Because of the anatomic structure and relationships of the esophagus, the highly malignant carcinomas of the organ, the frequent occurrence of metastasis, the danger of fatal infection, and the mutilation which must always cause great shock, surgical removal of these tumors cannot be expected to be attended with any degree of success."¹ This pessimism has proved in error so far as it pertains to the actual technique of resection. Despite the fact that most patients with esophageal carcinoma are elderly, and generally debilitated, the operative mortality of esophageal resection, though appreciable, is no longer forbidding.

The distressing fact is that the increased facility in thoracic surgery and all of the adjuvants available today have changed the picture of esophageal cancer only for the immediate postoperative period. The survival rate beyond a few months is still miserably poor, and cures at any given clinic are spoken of in terms of individual cases and scarcely in terms of percentages. Sweet² reports one five-year survival of 30 patients with resected carcinomas of the mid thoracic esophagus and Parker and associates³ report a group of 117 cases of esophageal cancer of whom only 2 patients were still living, both less than one year after operation. At the Johns Hopkins Hospital there have been no five year cures.

We are accustomed to emphasize to medical students that cancer is not primarily a mechanical disease and that its rational treatment properly should not be mechanical, i.e., surgical. Yet up to some point in the life of every cancer it is possible to cure the patient by excising the primary tumor with surrounding tissues. In today's stage of surgical virtuosity almost any viscus or combination of viscera can be successfully removed, if a few primary organs are left behind. However, surgeons tend to lose sight of the puerility of the concept of surgical treatment for cancer, and continue to devise more and more extensive operations for more and more hopeless lesions.

We propose an inventory of the present situation in cancer of the esophagus and an assessment of the results in an attempt to determine what should be the surgeon's orientation.

The posterior mediastinum can be compared to a conduit containing a number of axially directed structures closely apposed, and sharing lymphatic trunks

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and nodes. The esophagus lies in close relationship to the trachea, aortic arch, left bronchus, pericardium, descending aorta, diaphragm, and both mediastinal pleurae.

A cross section of the mediastinum of a full term infant (Fig 1) gives one some idea of the hopelessly intimate relationship of the esophagus with other structures.

The lymphatic drainage of the esophagus is both cephalad and caudad (Fig 2). Cephalad the drainage is to the bronchial and tracheo-bronchial nodes to nodes anterior to the aortic arch, and to nodes in the hollow of the arch.

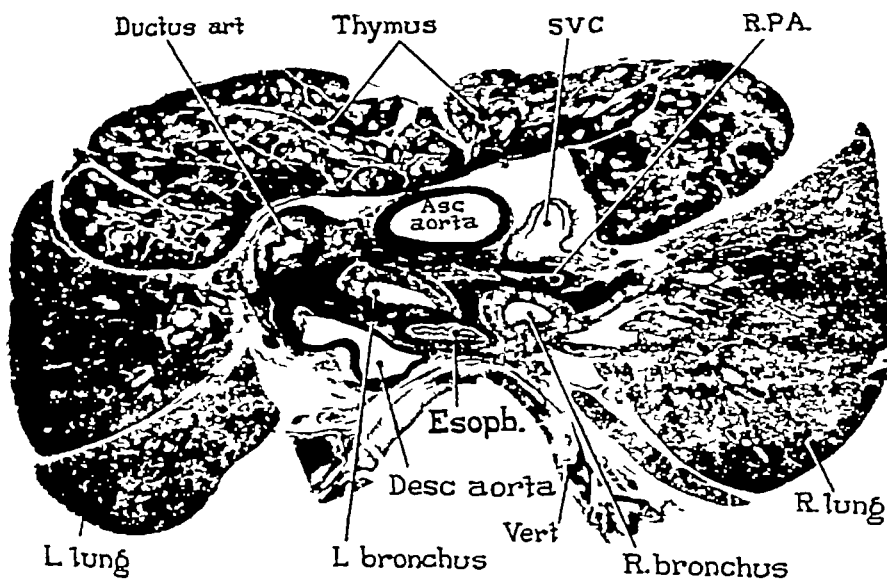


Fig 1—Cross section of thoracic viscera and vertebra of full term stillborn infant, anterior view. (Prepared by Dr J. R. Williams and Mr. Edw. Walker.) The section has been made just below the level of the bifurcation of the trachea. The extreme intimacy of the anatomical relationships of the esophagus is well shown. The esophagus nestles between the aorta and the left bronchus, is almost equally close to the right bronchus and lies immediately upon the vertebral body. With growth of the body the closeness of apposition scarcely changes and one readily sees how impossible it is to effect a radical resection of the esophagus. Of interest is the thick walled muscular still patent ductus arteriosus.

There are wide intercommunications between these several groups of nodes and with those draining the parietal pleura. Caudad the flow is to the nodes at the esophago-cardiac junction and the associated anastomotic pathways. Resection of the lymphatics would therefore require en bloc removal of both mediastinal pleura, nodes along both sides of the trachea and at the bifurcation, and those behind and in front of the aorta—a manifest impossibility.

The nature of the esophagus and its neighboring structures is such that resection, except perhaps at its ends, is it feasible to perform a block dissection taking a reasonably wide mass of tissue in all directions. One can hardly expect that simultaneous resections of trachea or bronchi will shortly be more readily performed than they are at present but even this is likely to be

in the nature of surgical gymnastics rather than a real advance in therapy with wide applicability. In desperate situations in the surgery of cancer it is customary to advocate more extensive operations. With the esophagus this can partially be accomplished by extending the length of the cylinder of re-

TAB XXI

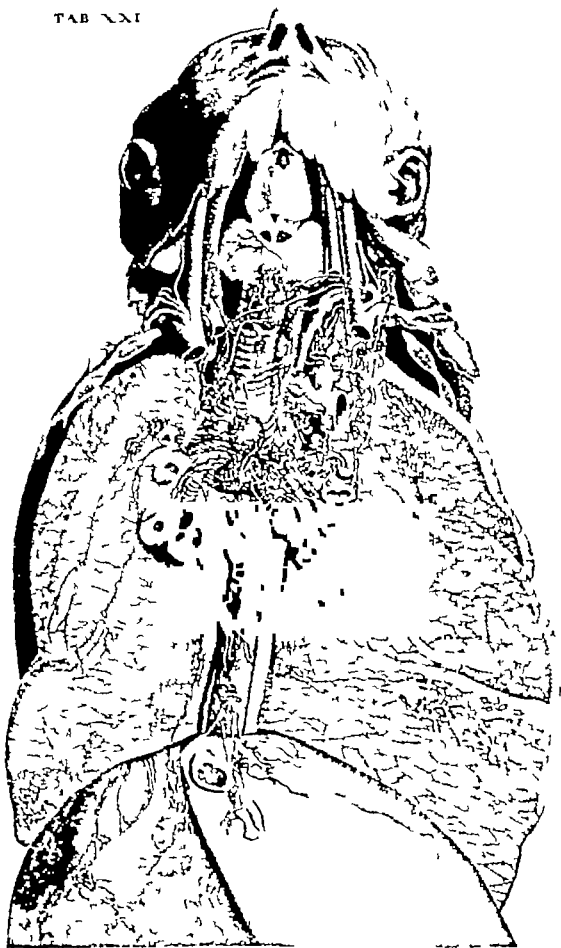


Fig. 2.—The lymphatics of the posterior mediastinum from the *Vasorum Lymphaticorum Corporis Humani Historia et Ichthyographia* of Paulo Mascagni published in 1787. More modern anatomical studies appear to have added little. Note the extensive communications of the bronchial, tracheobronchial, tracheal and paratracheal nodes with those of the esophagus; those anterior to the aortic arch and those in the hollow of the arch as well as the caudal pathways to the diaphragm and gastric cardia. Not shown are the nodes in the pleura and posterior parietals. Observe again the hopelessly disadvantageous position of the esophagus.

section. However, the diameter of the resected cylinder cannot be increased as long as one must leave behind other, closely associated, axially directed structures.

It might still be possible to cure cancer of the esophagus surgically in spite of these facts if it could be diagnosed early in its life history. But gloomy reports attest to the blind insidiousness and deceptive innocence of

the early symptoms, or the complete absence of any symptoms before even gross inoperability may be manifest. In addition esophageal cancer metastasizes early to the lymph nodes both cephalad and caudad. Furthermore, distant metastases are not uncommon. Possibly on this account patients with small, movable presumably early lesions are as likely to die of recurrence as are those patients with longer histories and larger tumors.

Thus, the evidence of the anatomical relationships of the esophagus which enforce a cylindrical resection, the mode of lymphatic spread which is outside the cylinder and the difficulty in obtaining patients early in the disease lead to the expectation that the curability of this disease will be low.

Actually Sweet² reports only one patient alive of 30 in whom the mid-thoracic esophagus was resected 5 or more years before. His results are somewhat better for carcinoma of the lower esophagus and cardia. Resano³ lists one 5 year and one 6 year survival of 1 000 patients treated on the service of Imochietto. Numerous similar reports of smaller series could be cited. Although direct operative attack offers one of the best methods of palliation it is associated with a greater mortality rate than less sizable procedures and gives little more prospect of cure. This method, then, leaves something to be desired and it is proper to consider the alternatives.

There is no need to review the dismal story of the improperly named palliative gastrostomy which has justly been generally abandoned.⁴ No form of therapy is adequate which does not rapidly restore deglutition.

In special cases—desperately debilitated patients, patients with severe cardiac or other complicating lesions—no type of operative procedure may be feasible. In these palliation can be achieved in one of three ways. Simple bougienage will often permit a patient to swallow fairly well for weeks at a time and can be performed repeatedly, but obviously without any influence on the outcome.

The Souttar tube, the use of which originated when the operation was almost invariably fatal, still has its place as Allison and Borrie⁵ have reminded us. We have occasionally employed a plastic variant which is forced into the malignant structure by pressure on a removable obturator. In one instance when thoracotomy revealed an inoperable lesion the tube was passed while the chest was open. This proved to be our most successful use of this type of indwelling tube. There was immediate restoration of deglutition and relief of the obstruction until death a few months later.

A third nonoperative method of palliation offers considerably greater therapeutic possibilities. Irradiation for cancer of the esophagus can be administered by radium bougie, by direct implantation of radioactive material, or by deep roentgen irradiation of various voltages. The most important and, it is observed in the course of the radiation therapy of cancer of the esophagus, is the usual rapid relief of dysphagia.⁶ In the majority of cases the patient is able to swallow for the duration of his course even if he dies of his disease.

Deep x-ray therapy, like operative extirpation, has not appeared to affect the outcome of the disease seriously except for individual instances of long term survivals. The radium bougie and the local implantation of radioactive material have in general not given as good results as has deep x-ray therapy. In a review of over 800 patients treated by irradiation, Fleming⁹ found that 83 per cent died within a year and that only 10 patients survived more than five years, one of these ultimately dying of metastasis. The results of the theoretically superior rotation treatment appear to be comparable.¹¹

Of great importance is the observation that after irradiation of esophageal cancer some deaths occur from perforation into the mediastinum, pleura, or aorta and at autopsy no residual tumor is found. This suggests the possibility that if before irradiation the esophagus were removed or rendered empty and clean by short encircling, such deaths might be obviated. In this connection a case has been reported by Lincoln Brown¹² in which at operation a silver tube was passed into the esophagus and a 5,000 roentgen tumor dose of x-ray subsequently administered. No tumor was demonstrable endoscopically 6 months later.

When cancer of the esophagus occurs in a patient who is a reasonable candidate for operation, circumstances may indicate one of several procedures.

It has been demonstrated that the stomach can be mobilized so as to permit a primary anastomosis to any level of the esophagus, even well into the neck. However this procedure is a considerable task, requiring extensive dissection and for the high tumors a difficult anastomosis. On the other hand for tumors of the esophagus in the lower fourth, resection and esophago-gastrostomy is to be recommended when the tumor is resectable. If a low esophageal carcinoma or carcinoma of the gastric cardia cannot be resected the obstruction may be by-passed by dividing the esophagus, closing the lower end and connecting the upper end to a Roux-Y jejunal loop. Only one or two jejunal vessels need be sacrificed to prepare such a loop, its circulatory adequacy need not be a cause for concern, and the operation is readily performed.

In the higher tumors there is some reason to doubt the advisability of elevating the stomach. In addition to the technical hazard of the high anastomosis there is the disadvantage of the presence of a distensible stomach in the thorax. Regurgitation of gastric juice into the pharynx is often a source of distress with the added hazard of aspiration. The risks of an intrathoracic anastomosis of a very long jejunal loop seem unjustified,¹³ but an antethoracic Roux-Y loop can be mobilized with much less difficulty than that attendant upon mobilization of the stomach and with little more risk to life than that inherent in a small bowel anastomosis.

In the day of the Torek procedure numerous attempts and some successes were recorded in reconstructive antethoracic esophagoplasty. Stomach, jejunum, and colon were all employed as well as the several varieties of epidermal tubes and combinations of skin and intestinal tubes. This frequently entailed a multiplicity of procedures requiring many months. As a result ante

thoracic esophagoplasty was held inapplicable to the problem of reconstruction after resection of cancer of the esophagus with the anticipated relatively brief survival. This attitude can be revised now in the light of greater experience with esophagoplasty as demonstrated by the work of Yudin¹⁶. Greater familiarity with the technique has reduced it to a two stage operation with a delay of only a few days between stages.

The operation reported by Roux¹⁷ in 1907 for constructing an antethoracic esophagus or jejunum is the method of choice in antethoracic esophagoplasty. In most patients the jejunum can be brought above the clavicle and anastomosed to the esophagus a few days later. It is wiser, rather than to risk a slough, not to attempt to pull the jejunum higher than it will go readily. In two of our cases resection of the medial end of the clavicle permitted tension free anastomosis of jejunal tube and esophagus. Three and at times four, principal jejunal vessels may be divided to provide a long Roux-Y jejunal loop. All serosa is excised from both sides of the mesentery of the loop which is brought through the mesocolon and subcutaneously. A wide subcutaneous tunnel is made from the xiphoid to the neck and the bowel is then gently pushed and drawn through it. A temporary feeding gastrostomy is constructed. Anastomosis with the esophagus is accomplished several days later. Roux anastomosed the lower end of his jejunal loop to the stomach. Two experiences in young adults who developed peptic jejunal ulceration have led us to distrust this step. There has been recurrent discussion of the advisability of the use of an antiperistaltic jejunal loop which is thought by some to be easier to raise^{18, 19} although the published experiences have been unsatisfactory.

There are many patients who will tolerate a Torek procedure but who would not be likely to survive the additional strain of mobilization of the stomach and the interference with pulmonary function caused by placing the stomach in the chest. Antethoracic esophagoplasty may be performed as soon as such a patient is well over his thoracotomy and he may be expected to be taking his food orally within another two weeks. Small fistulae at the site of the cervical anastomosis are not rare but usually close spontaneously.

This therapeutic approach has the following advantages:

1. There is no tendency to compromise in the amount of esophagus resected and whatever virtue resides in resection is reserved to the patient.
2. The major intrathoracic procedure is short and relatively simple.
3. The anastomosis is extrathoracic and a relatively innocent procedure as far as concerns risk to the patient or effect upon his general condition.

There is an appreciable risk in elderly arteriosclerotic patients, of losing a portion of the oral end of the jejunal loop if it is made long enough to reach the cervical esophagus. When an antethoracic shunt is employed a portion of the tube may slough in the subcutaneous tissues as in two of our patients, without causing any major distress. One patient with desperate coronary disease succumbed before the esophagus could be completed but the Roux-Y loop offered him a satisfactory leak proof feeding jejunostomy. In the other

patient the antethoracic esophagus was ignored after the proximal end sloughed. The inoperable tumor was partly resected and esophageal continuity successfully restored by use of a plastic tube (*vide infra*). It may be impossible in adults, with sclerotic vessels and thick fat mesenteries, to raise loops to the neck as often as in children. However, as in Roux' own case the cervical esophagostomy may be connected to the antethoracic jejunostomy by a skin tube. Such a skin tube is fashioned and anastomosed in a single stage. If the skin of the chest wall is particularly lax the tube made of the thoracic skin may be covered by loose skin drawn from the sides. If such skin is not available the tube may be covered with a free split thickness graft. The tube itself is so fashioned as to include and enclose, at upper and lower ends the esophagostomy and jejunostomy.

In 1922 Neuhof and Ziegler²⁰ experimented in dogs with the use of rubber tubes as replacements for esophageal segments. In animals they were regularly successful with a two stage operation. In the first stage the esophagus was packed off to produce granulation tissue and seal off the mediastinum. In the second stage the esophagus was resected and a rubber tube tied into the upper and lower segments. The tube usually passed after a time and the resultant esophagus was found to be lined by normal appearing mucosa, to be strong and smooth, and to show no tendency to obliterative cicatrization.

In dogs, with then thin pleura, Neuhof was obliged to work with the cervical esophagus but felt that in patients the two-stage operation could be applied to the thoracic esophagus approached extrapleurally through the posterior mediastinum. Berman of the Sinai Hospital, Baltimore, has developed a method for replacement of the thoracic esophagus with a plastic tube. First in experimental animals²¹ and then in a series of patients he demonstrated the feasibility and simplicity of his method. The desired length of esophagus is resected a large calibre flexible, flanged Polythene tube is sewn into the upper and lower ends and tied in place, the mediastinal tissues being then closed about the tube. In our clinic five such operations have been performed by four surgeons. In four instances the operation was performed without difficulty and the patient subsequently swallowed well, though in one patient the tumor was grossly inoperable and cancerous tissue was left on the aorta. One is still in the hospital gravely ill, but able to swallow well. All of these patients were operated upon through the right side of the chest. In a fifth patient, operation was performed through the left chest and the tube placed anterior to the aortic arch with fatal outcome.

Although one may have an a priori objection to the use of buried prosthetics in surgery, it would appear that, in this instance the tube acts only as a stent about which fibrin forms an impermeable sheath which is then lined by esophageal mucosa growing in from either end. Barium swallowed a week after operation passes through the lumen of the tube and may pass partly between the outside of the tube and the encasing mediastinal tissues. Although Berman has employed tubes of considerable length there has appeared in practice an understandable tendency to skimp on the length of



FIG. 1. W. This is a 75-year-old man with a three-month history of dysphagia and weight loss. The radiograph shows a marked constriction of the middle third of the esophagus. Because of the marked constriction and the apparent extensiveness of the tumor, an attempt was made to perform a 15-cm stricturoplasty. The procedure was performed. He died four months later. The specimen is shown in this photograph and takes a semisolid consistency. A 15-cm specimen showing the constriction and irregularity of the tumor. The specimen is shown in this photograph.

esophagus resected in order to make the use of the tube feasible, particularly when the carcinoma is near the apex of the pleura. The use of this plastic tube provides the quickest and easiest method of esophageal reconstruction and simplifies the operative attack on esophageal cancer. The use of the tube is most applicable to carcinomas of the mid portion of the esophagus.



Fig. 4—C. C. Postoperative roentgenogram of a 37-year old woman operated upon April 14, 1952, by Dr. William F. Rlenhoff, Jr. A carcinoma of the middle third of the esophagus was resected and the defect bridged with a polythene tube which extended from the arch of the aorta to the diaphragm. The x-ray shows barium and air in the plastic tube.

SUMMARY

The results of the operative treatment of carcinoma of the esophagus are disappointing in terms of survival and of cure rates. The organ is fundamentally so situated, and the disease of such a character, as rarely to lend itself to cure by operative means. It seems justifiable therefore to direct our efforts toward palliation rather than cure and to seek the methods which will yield the best return in terms of patient months of comfort, with deglutition restored.

In manifestly incurable tumors swallowing may be restored by irradiation, or by a shunting procedure, preferably antethoracic esophagoplasty. In cancer of the lower fourth of the esophagus resection and esophagogastrostomy is the treatment of choice for resectable lesions and an intra thoracic shunt with a

Roux-Y jejunal loop the treatment of choice for inoperable lesions. In the mid esophagus three operative procedures are available for resectable tumors each with its own advantages:

- 1 Resection and esophagogastrostomy
- 2 Resection and antethoracic esophagoplasty
- 3 Resection and plastic tube reconstruction

For nonresectable tumors and in debilitated patients antethoracic esophagoplasty may be offered with supplemental irradiation.

Lesions near the apex of the thorax are best treated by antethoracic esophagoplasty with or without resection or irradiation.

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DISCUSSION

DR J F MAC MANUS, Buffalo—We have had some experience with Dr Berman's technique of esophagectomy in the upper third of the esophagus. Our series numbers five cases. We have had no operative mortality. I thought you might like to

see a specimen. This patient lived four months after surgery. We have this post mortem specimen, and I would also like to show a cineradiographic picture demonstrating the method of passage of food through this type of prosthesis.

(Slide) This demonstrates the pathology in this case. The operation is not prolonged, not difficult, and even patients who are poor risks will tolerate it rather well.

(Slide) This is the polythene tube lying in the mediastinum with the distal and proximal part in view, and the lower end of the tube. The picture was taken approximately three weeks postoperatively. There is some lipiodol which he is not able to force through this opening that escaped down into the trachea.

(Slide) This picture was taken after the dye had gone down, and some of it has become adherent to the polythene tube.

(Slide) This is the specimen four months after surgery. There is a solid tube of carcinoma and fibrous tissue, very tough and hard, surrounding the polythene tube. The patient was swallowing a liquefied and soft diet satisfactorily.

(Movie) This is a cineradiographic picture taken of this man six months after operation. It runs a circle, keeps repeating itself about every eight seconds, so that you have to watch it more or less as you would a football game, first the left end then the center next, etc. Otherwise, if you try to look at it all at one time you miss a good deal of the detail. As you watch this you see the barium coming down in and around the tube as well as through the tube.

DR LYMAN A. BREWER, III, Los Angeles—We have had disappointing experiences in curing carcinoma of the esophagus similar to those presented by Dr Ravitch. The forms of palliation which were so nicely shown by the speaker have an important place in treating definitely inoperable carcinoma of the esophagus. It is regrettable that the majority of the cases seen by surgeons today fall into this category. However, since surgical resection of the esophagus is curative when the cancer is confined to the esophagus, we strongly believe that more strenuous attempts should be made to diagnose the carcinoma in the early, resectable stage. The high mortality is linked with operations on advanced cases in most instances. Unfortunately, the textbooks of medicine generally present only advanced findings for the diagnosis of carcinoma of the esophagus, and the important early signs and symptoms are usually not mentioned.

The early symptoms are those of mild discomfort, soreness, or tightness in the thorax or upper abdomen. If the esophagus is considered, along with the lungs and heart, as a possible cause of these mild symptoms, then the early cancer will not be overlooked. It cannot be stressed too much that *at first the dysphagia is not progressive*. There are times in the early stage of the cancer when, for various reasons, the patient swallows better. The carcinoma acts somewhat as a foreign body, with resultant associated esophagospasm. When the esophagospasm associated with an early carcinoma of the esophagus is relieved by belladonna preparations, then both the physician and the patient are lulled into a sense of false security by the belladonna response. At least one third of our cases have shown this. Sloughing of the tumor will also permit the patient to swallow better for a while. The frequency of reference of symptoms caused by carcinoma of the esophagus to the abdomen focuses the attention of both physician and patient upon abdominal disease. Most often a careful study of the stomach and intestines is made, and examination of the esophagus is overlooked. We have had a number of cases in which the proper diagnosis could have been made readily from six months to one year earlier by thorough esophageal study when the symptoms were primarily epigastric in nature. Furthermore, the cancer of the lower esophagus that arises in the cardia of the stomach presents diagnostic problems. Since this type of carcinoma spreads to the esophagus in the muscularis and submucosal layers, the mucosa shows no tumor or ulcer. However, if an organic stricture of the esophagus is present at the cardia, or there is an inelastic infiltrated muscularis, then this patient should be explored.

It is certainly the duty of the thoracic surgeons to teach the pitfalls in the early diagnosis of cancer of the esophagus and in the future, we will have many more cures to

report. We have been told that the original case of esophageal resection for carcinoma of the esophagus with esophagogastronomy, performed by Adams and Phemister in this country in 1928, is still alive. The long time survival of this one patient should spur us all to teach the early diagnosis of carcinoma of the esophagus.

DR OWEN H. WANGENSTEEN, Minneapolis—I can sympathize with Dr Ravitch in his statements concerning the difficulties of esophageal cancer surgery. It is now almost ten years since Ohsawa in Kyoto, Japan, first announced the successful reestablishment of continuity after excision of cancers of the fundus and cancer of the lower esophagus. It seems a bit strange that after all these years we have not done better. The only five-year results that have been published to date are those of Richard Sweet, and, as he said in his recent article in *Surg., Gynec. & Obst.* (94:46, 1952), the results of surgery for cancer of the mid and upper esophagus have been so poor that he hesitated to record them. However, you will recall that in his operations for cancer of the lower esophagus and the fundus and of the stomach, with patients not having lymph node involvement, 40 per cent of the patients were alive after five years. The tragedy is, of course, that so many do have lymph node involvement, as I recall it, his figures for those having lymph node involvement showed five-year survival in 15 per cent of the patients.

Julius Caesar propounded an old military maxim when he said, "Divide and conquer." This is true in military as well as in surgical matters, and I am not referring primarily to the procedure—on the contrary, I am referring to dissection of the problem. I too have suffered from the experience of losing patients for no obviously good reason following excision of cancer of the mid and upper thoracic esophagus. Sometimes the loss of the patient is very difficult to understand, there being at autopsy scarcely anything to account for the fatal outcome. The most common finding appears to be atelectasis. In thinking this over two thoughts occurred to me. Shortening the path of the migrated stomach permits excision of a greater length of esophagus on the proximal side of the lesion. This is certainly an important objective in curing cancer of the esophagus. It is desirable to excise five to six centimeters above the cancer. The shortest path for the migrated stomach is through the right diaphragm, anterior to the left lobe of the liver and anterior to the pulmonary root—that is, anterior to the lung. This path seems to be several centimeters shorter than the conventional path through the hiatus and posterior to the lung root.

But what causes the atelectasis observed in these patients? Well, mismanagement of the fluid problem can do it by inviting overhydration. However, I think the main thing is reanastomosis into the pharynx and the larynx from a very short residual esophagus. One thing which I have done hitherto which has made some difference in patients recently operated upon is to leave the indwelling intratracheal tube in place until the patient is very much awake. In the meanwhile in the recovery room the nurse aspirates the pharynx and mouth until it is quite dry. Then when the patient wakes up, the cough reflex will be sensate materially the chance of aspirating mouth or gastroesophageal secretions into the trachea. The other thing which I have come to employ, and which I believe has some bearing on the occurrence of atelectasis, is placement of a No. 14 French hole duodenal tube into the jejunum at the duodenojejunal angle. This tube is threaded backward into the thoracically migrated stomach. This catheter can be left in situ well into the convalescent period. And finally the application of suction to this tube has given considerably the opportunity for aspiration of gastric juice or bile into the trachea. A No. 14 French hole duodenal tube is inserted into the gastric pouch from above by the anastomosis when the esophagogastronomy is completed. This tube need only remain in a few days. However, during the waking up period, by retaining the intratracheal tube until the patient is fully awake and the avoidance of reflux into the trachea during the waking up period—these two expedients I believe will be found very effective in preventing atelectasis and thus attention to excision of cancer in the mid or upper

esophagus is largely up and down the reaches of that. It is very important to try to excise long length both above and below, most

of us having compromised too much on the excision above the cancer, being satisfied with a margin of a centimeter or two. This is not enough. For cancers in the lower esophagus, I have come to divide the azygos vein, anastomosing above it, routinely. I have seen no suggestion that placement of the stomach in front of the lung invites atelectasis.

Placement of water filled rubber balloons on top of the pulmonary root in dogs, that walk on all fours, does not invite atelectasis. The factors hedging about the item of aspiration certainly are far more important.

In patients under 50 years of age with an active gastric secretion having an esophageal cancer, I am not certain that the stomach is the best replacement organ for the esophagus. The only good thing to say for it is that it is readily available. In such instances, the colon is probably a far better organ, though the technical difficulties in employing it rather than the stomach are obvious. In patients with an active gastric secretion, any extra length of stomach, not needed at the upper end, should be excised. The observations of Demel (*Langebeck's Archiv* 128: 453, 1924) have important connotations for resection of this segment of the esophagus for cancer, suggesting that the mid and upper thoracic esophagus do not have as rich an arterial blood supply as the cervical and terminal esophagus.

Whereas there may be an occasional indication for the type of procedure which Dr Ravitch has outlined, I would like to suggest that it is too early to capitulate to palliative procedures. We must continue our dissection of, and our attack upon the problem. If we can learn to master the intricate technical considerations and the potential physiologic hazard which hedge about the situation, after excision of long lengths of the esophagus, the initial operative mortality will be far less formidable. Such has been the history of all operations. And, if greater lengths of the esophagus are sacrificed, the number of ultimate cures will also be greater. X-ray irradiation is still not a strong competitor for the high thoracic living esophageal cancer. Continuance of efforts at surgical cure is therefore still in order, even though the risks are large and the present cure rates are low.

DR OSLER ABBOTT, Atlanta—I am sure that the factors mentioned by Dr Wangensteen are of utmost importance. There is a contribution which my associate, Dr William Hopkins, has made in the last few years which is in the process of being presented. I think this work designates his understanding and appreciation of the physiologic disturbance of transection of the diaphragm. I think that is of major importance. The anatomic distribution of the phrenic nerve, as it comes across from its point of insertion anteriorly and medially, must not be interrupted in order to maintain the optimal function and maximal activity of the diaphragm postoperatively. Therefore, Dr Hopkins recommends that the diaphragm be incised circumferentially about 2 to 3 mm inward from its costal attachments. This gives a remarkable access to the abdominal contents. Furthermore, if the pathologic process does not require excision of the area of the hiatus, the hiatus and the crura muscle, so intimately appreciated by Dr Alison, are maintained intact. This preservation of diaphragmatic function, both for respiratory processes and for the pump action of the crura muscles, is of distinct value in preventing unnecessary postoperative debility and complications. It emphasizes that physiologic considerations in major and so called minor surgical details are the prophylaxis in regard to postoperative discomforts.

DR ROSS ROBERTSON, Vancouver, B. C.—I agree in part with Dr Ravitch and Dr Wangensteen, but instead of taking Dr Ravitch's high road beneath the skin, or Dr Wangensteen's low road through the left chest, I choose to take the middle road through the interior mediastinum, through which to bring the Roux-Y tube. Since reporting on reconstruction of the esophagus here two years ago, we have had eight additional cases of reconstruction for carcinoma of the esophagus. Two of these patients died postoperatively. One was a man aged 77 years, the other man was riddled with secondary tumor. Three others are still alive, two to ten months after operation. The remaining three survived from seven to twelve months after operation.

Of the survivors, all were able to swallow quite readily and with considerable satisfaction postoperatively. I have come to the conclusion that probably the way to treat car-

carcinoma of the esophagus may be summarized as follows. If a carcinoma in the lower quarter of the esophagus is squamous-cell carcinoma not arising in the stomach, I think Dr. Sweet's method is preferable, that you have an opportunity of resecting the entire carcinoma and not getting recurrence at the anastomosis. In all other carcinomas of the esophagus the Roux Y tube should be prepared, brought up through the anterior mediastinum and the patient enabled to swallow with comfort.

Then if one feels that the tumor may be resectable, he may go back two or three weeks later through the right chest and remove the entire esophagus for several centimeters on each side of the tumor, or the entire stomach if necessary and all the surrounding lymph nodes.

DR JOHN H GIBBON, JR, Philadelphia. It seems to me that this Association has in the last couple of years a tendency toward backing away from attempts at radical cur of carcinoma because of a rather forbidding mortality. Some of us had a discussion the other night about the same subject relative to carcinoma of the lung. It seems to me that there is another attitude to take, that is, to continue with an aggressive attitude in the attempt to eradicate the disease, and to devote our efforts toward trying to find out the cause of these unexplained deaths that Dr Wangenstein mentioned. I appreciated very much Dr Ravitch's philosophical and historical discussion, but I must differ with him in my attitude toward the philosophy. I am about to reverse what I said three years ago before this Association. About nine months ago Dr Albritten, Dr Templeton, and myself decided that we would reverse ourselves about the left-sided approach and use a right thoracotomy for esophageal lesions at the level of, or cephalad to the aortic arch. We hoped to lower the mortality in the higher esophageal lesions, which is the real crux of the problem. In the last nine months we have operated upon five patients with esophageal lesions at the level of the arch or in the neck with one death, using a right thoracotomy.

I would particularly like to advocate the use of two operating teams for lesions of the cervical esophagus or the thoracic inlet. With two operating teams, you can cut the operating time in half. One team mobilizes the stomach in the abdomen while the other team mobilizes the cervical esophagus and performs when indicated a radical neck dissection with or without a hemithyroidectomy or laryngectomy. Then with the patient still in the supine position, the right chest is opened by the team operating in the neck. The thoracic esophagus is mobilized and the abdominal operating team passes the stomach up into the thorax. The other operating team passes the stomach into the neck and performs the esophagectomy there while the abdominal team closes the abdominal and thoracic incisions.

I think we should devote still more time to the aggressive attack on these lesions with an attempt to reduce the mortality rather than to throw up our hands and say that this is beyond us. We have recently reviewed our results in 148 patients with cancer of the esophagus or of the gastric cardia. This is a consecutive series seen in the past six years. The lesion was resected and an anastomosis performed in 91 of these patients. From this group in addition to Dr. Ravitch's five year survivals one case. Of course the majority of our cases have been seen in recent years, not five or six years ago. In conclusion I think it is probably if we continue our effort toward reducing mortality and keep up an aggressive attack upon the disease.

Dr. FAVITCH (Chicago).—Dr. Mc Manus mentioned his use of the Berman tube with its use of continuous suction. Dr. Berman tells me it is not necessary to drain the mediastinum. None of the cases at Baltimore of which there have been 19 at various hospitals treated by Dr. Berman's own party had the mediastinum drained and of the 19 there has been only one fatal result.

I have been disappointed if Dr. Wangerman and Dr. Gibbon had not
with a further set of views. I think perhaps we will have to go back a little
of the structure to the architect who placed the esophagus in such an awkward
position. Dr. Wangerman is a little better than we are. I am sure that
we cannot find a solution by any other way than the lower portion of the esophagus
being placed in the center of our cavities and then bringing it up either

intrathoracically or mediastinally. We have had experience with two patients in whom there was a subcutaneous slough. One is a man who has had many attacks of coronary disease, and he sloughed off several inches of the antethoracic esophagus. It left him a beautiful feeding jejunostomy as a result which should not leak at all, and, incidentally, one of the things a Roux loop will give is a beautiful feeding jejunostomy anywhere, without leakage. The other patient had a slough of the entire antethoracic loop, to the costal margin was not sick at all, and his surgeon elected then to resect the tumor and place the plastic tube in situ, although he had to carve the tumor from the bronchus and the aorta. He got by in that instance.

I would like to say again that while we do not class ourselves as among the timid group of surgeons, the point is that the esophagus is not like the breast which is virtually a skin appendage, practically off the body, and one can do a well conceived cancer operation there. It is just not possible to take out the esophagus that way.

BRONCHIOGENIC CARCINOMA A PRODUCT OF MULTIPLE SITES OF ORIGIN

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A LARGE segment of opinion has long held to the principle of the unicellular or unicentric origin of neoplasm. Under the influence of Cohnheim,¹ Mallory,² McFarland,³ and Kettle,⁴ to mention only a few, this hypothesis has been widely accepted. Others, however, have from time to time disputed its validity. In recent years Willis⁵ has gathered a formidable body of evidence indicating the origin of tumors from multiple sites and has, himself,⁶ provided convincing data supporting the earlier work of Lohmer⁷ in relation to tumors of the lip and skin. Many similar observations⁸⁻¹¹ have served to support the principle of the multicentric origin of neoplasm in other areas.

Save for the diffuse form of so called alveolar cell carcinoma, however, little attention has been directed to this phenomenon in the case of pulmonary neoplasm. Isolated case reports have cited instances of multiple gross and microscopic primary carcinoma in lungs¹²⁻²² but only in the meticulous study carried out by Lindberg²³⁻²⁴ has there been evidence of a more than sporadic incidence. One suspects that failure to observe this phenomenon as a common one may be attributed in large part to the custom of relying upon sections from small blocks of tissue removed directly from the presenting tumor and from obviously invaded lymph nodes. The possibilities inherent in the study of bronchiogenic carcinoma through the medium of large tissue sections were beautifully demonstrated before this Association in 1949 by Phillips, Adams and their associates.²⁵⁻²⁸

Since 1947 the Departments of Surgery and Pathology at the University of Cincinnati have been engaged in a series of studies utilizing such large sections. A recent investigation of carcinoma of the stomach²⁹⁻³¹ by this means revealed an unsuspected frequency of multiple neoplastic zones and stimulated our interest in applying this technique to bronchiogenic carcinoma.

MATERIAL AND METHODS

Over 100 gross specimens of lung were processed from a number of hospitals in the Greater Cincinnati area.³² Of this group 94 contained neoplasm.

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In 4 instances the lesions were bronchial adenomas, in 1 there was a hamartoma, and in 2 the processes were diffuse lesions of unusual nature. These were excluded, leaving 87 specimens which were utilized as the basis for this report—all examples of indisputable bronchiogenic carcinoma.



Fig 1 A.—Bronchiogenic carcinoma occluding a bronchus in the right upper corner of the photograph. The upper lobe is atelectatic, the lower lobe the seat of severe bronchiectasis. The ink dots indicate the approximate extent (9.9 cm.) of a contiguous plaque of preinvasive carcinoma. ($\times 12$)

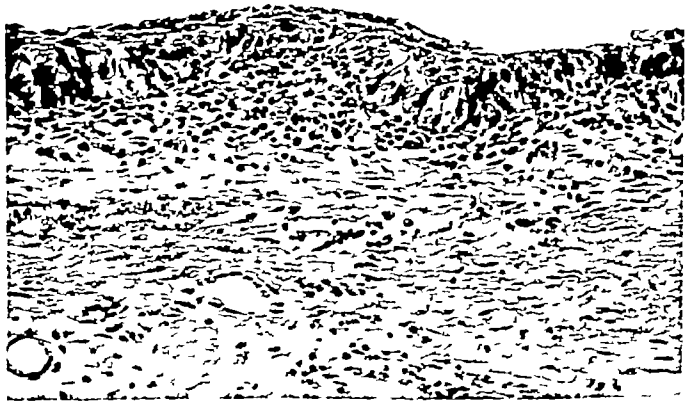


Fig 1 B.—High power view of the contiguous plaque of preinvasive carcinoma in Fig 1 A. Epithelial atypism and the intact basement membranes are noteworthy. ($\times 300$)

Fifty of the specimens were obtained at autopsy and 37 by surgical excision. The lungs were delivered for processing as soon as possible after removal and in effort was made to procure roentgenograms of the inflated specimens. Formalin, 10 per cent, was introduced into the major bronchus at low pressure, and the entire specimen was immersed in vats of this fluid until fixation was completed. Full length sections through the cancer-bearing lung were then prepared in

such a manner as to contain the bulk of the neoplasm as much as possible of the bronchus to which it was related, and adjacent nonneoplastic pulmonary tissue. The area of the block was limited to 8 by 12 cm by the size of the cover slips. If necessary the tumor was divided in order that a full-length section of the pertinent bronchus could be retained in continuity with the tumor. The number of such blocks ranged from 1 to 4 per specimen in the

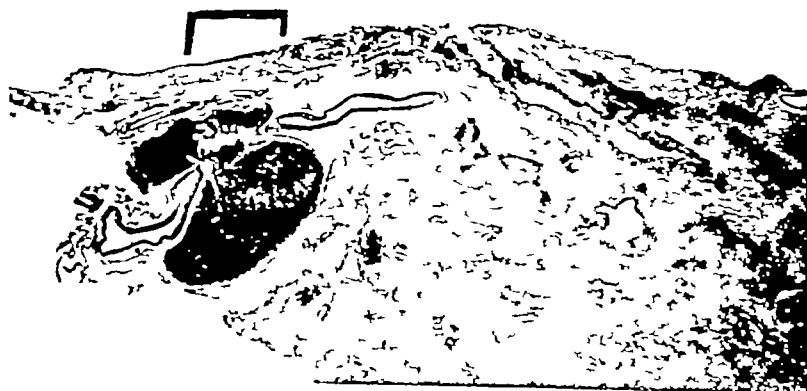


FIG. 1.—A collapsed lobe with neoplastic bronchostenosis in a secondary bronchus indicated by the two ink dots. Extending proximally in the area outlined by the bracket is a thickened plaque of preinvasive carcinoma measuring 1 cm ($\times 12$).

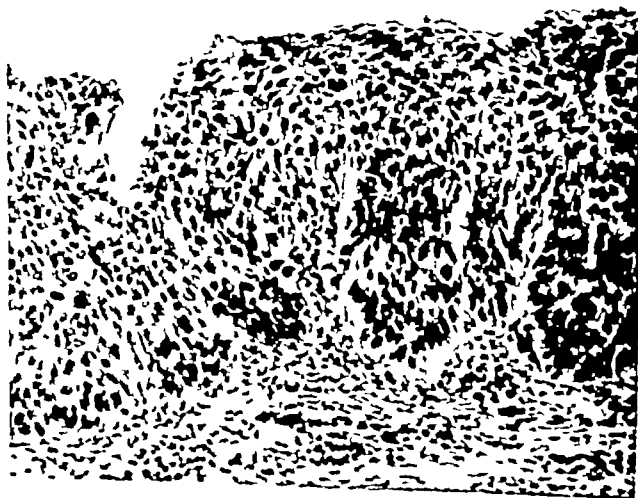


FIG. 2. Histology view of the continuous plug in Fig. 1. The neoplastic appearance of the tumor and the noninvasive character apparent ($\times 300$).

At least majority 2 tissue sections were prepared. In certain instances additional trapper samples were removed from areas which aroused interest and from these trapper samples of conventional size were prepared.

The tissues were embedded in paraffin and sections made on a large sliding microtome at thicknesses approximately 8 to 10 μ . Sections were uniformly stained with hematoxylin and eosin.

OBSERVATIONS

In the earlier study on the stomach²⁹ 3 microscopic features had proved to be of significant interest and the present investigation has been directed along similar lines. The features sought were (a) the presence of neoplastic alterations



Fig 3 A—Carcinoma straddling the bifurcation of a primary bronchus. The mucosa between the single ink mark and the bracket is nonneoplastic. The bracket outlines the position of an independent focus of carcinoma. ($\times 12$)

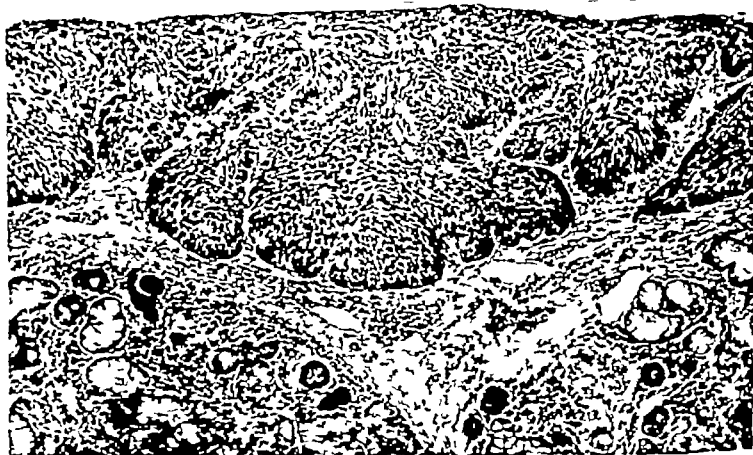


Fig 3 B—High power view of the entire extent of the independent carcinoma in Fig 3 A. Early invasion has occurred. ($\times 90$)

of microscopic character in the mucosa immediately adjacent to the carcinoma (b) the occurrence of independent foci of neoplasm of both gross and microscopic nature, and (c) the intratumor variations of histologic growth patterns

In 13 specimens the bronchial mucosa in immediate continuity with the main tumor was found to be the seat of premalignant squamous cell carcinoma (Figs 1 and 2). This lesion was characterized by metamorphosis of the normal bronchial epithelium into bizarre stratified squamous epithelium exhibiting nuclear atypism, mitotic figures in abundance, cellular pleomorphism, and loss

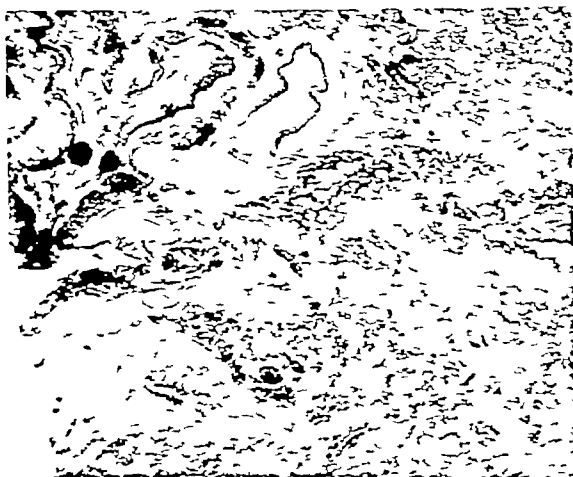


FIG. 1.—A partially collapsed lobe distal to an area of neoplastic bronchostenosis not shown in the photograph. In the upper left hand corner is the bifurcation of a bronchiectatic secondary bronchus filled with purulent exudate. The ink dots designate the location of two independent foci of neoplasia 3.5 and 5.9 cm distal to the major tumor ($\times 12$).

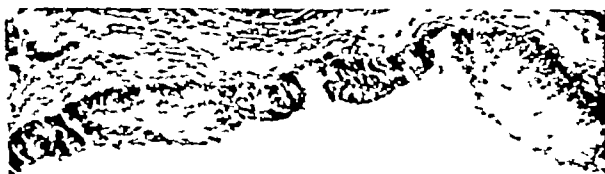


FIG. 2.—A section of the bronchus showing the transition between the normal bronchial epithelium and the neoplastic squamous cell carcinoma. The main focus of the carcinoma is located between the two ink dots.

of orientation. In general, the process was limited by an intact basement membrane (Figs 1,*B* and 2,*B*). Occasionally the alteration appeared also within the ducts of bronchial glands similar to that seen in carcinoma in situ of the



B

Fig 5 A—A segment of lung the upper bronchus of which is the seat of carcinoma. The ink dots represent the location of two independent foci of metaplasia 5.0 and 7.0 cm. proximal to the invasive cancer ($\times 12$)

Fig 5 B—One of the independent foci of preinvasive carcinoma shown in Fig 5 A ($\times 90$)

cervix of the uterus. Rarely, the basement membrane was breeched by superficial invasion, thus attesting to the malignant potentiality of the lesion. Because of the shelflike character of the process and its intimate relation to the main

tumor, we have designated the alteration as the contiguous-plaque phenomenon. When more than one margin of a tumor was included in sections, contiguous plaques occasionally appeared in more than one site. They ranged in breadth from 1 or 2 mm to as much as 7 cm beyond the obvious limits of the tumor.

There were also found in 19 specimens identical microscopic foci which, however, lacked continuity with the main neoplasm. Believed to be independent growth centers, these small plaques of preinvasive carcinoma were separated by varying distances of unaltered mucosa (Figs 3, 4, and 5). Indeed the intervening space ranged from 1 cm to as much as 10 cm and occasionally 2 to 6 such lesions were detected. Nor were they limited to the bronchus or major involve



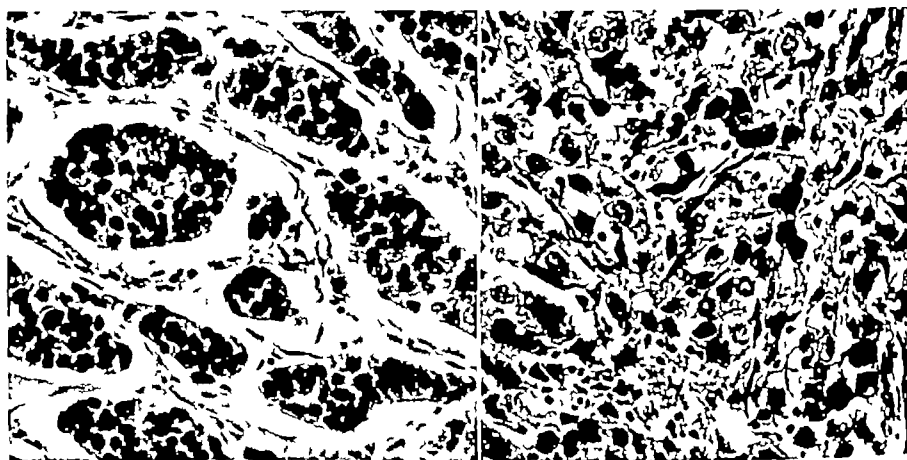
Fig 64.—A bronchiogenic carcinoma obliterating the lumen of a bronchus in the right upper corner. The ink dots indicate the areas in which different histologic patterns are seen.

ment, but appeared in subordinate branch bronchi both proximal and distal to the cancer and even in other lobes in which no neoplasm was suspected on gross inspection. Though usually of small extent (i.e., a single low-power field (Figs 3,B and 4,B)), some of these lesions measured up to 3 cm. As was the case with the contiguous-plaque phenomenon, shallow invasion was observed on occasion.

Both the contiguous plaque and the independent microscopic lesions appeared in the same lungs in 7 specimens. In 25 others either one or the other process alone was encountered. In 5 additional lungs 2 grossly visible independent carcinomas were observed.

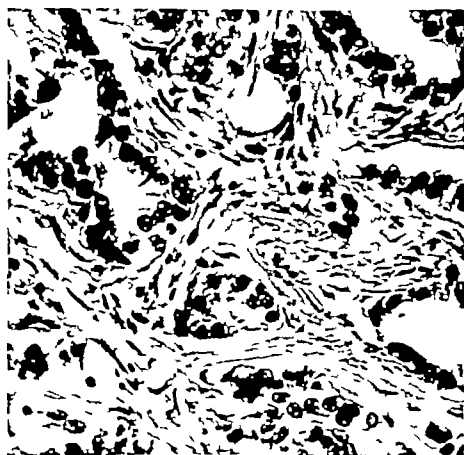
The value of the large section technique was particularly demonstrated when the histologic structure of the neoplasms was scrutinized. Although pleomorphism has been remarked repeatedly in bronchiogenic carcinomas,³⁰ the

degree actually observed in this series was most impressive. Less than half of the neoplasms (42 specimens) revealed only a single type of architecture throughout. In the remaining 45 cases, as shown in Table I, 116 different structural patterns were encountered. In many, of course, the variation might



B

C



D

Fig 6 B—High power view in region of an ink dot in Fig 6 A. The pattern here is that of a small cluster alveolar carcinoma. (All high power views in Figs 6 and 7 are taken at identical magnification.)

Fig 6 C—Another focus in Fig 6 A showing an undifferentiated nonorganoid pattern.

Fig 6 D—A third structural pattern in the tumor shown in Fig 6 A. Here the lesion is a well differentiated adenocarcinoma.

be considered to be one of degrees of differentiation of a single type of cell. But in a goodly number no possible developmental relationship could be attributed to the 2 or more patterns noted in a single tumor mass (Figs 6 and 7). In this category were combinations of well differentiated squamous cell car-

cinoma with equally well differentiated adenocarcinoma spindle cell alveolar carcinoma in association with oat cell carcinoma anaplastic giant cell carcinoma or even formless clusters of signet-ring carcinoma

There were 2 patterns evident in 27 tumors 3 in 16 cases and 4 or 5 patterns in 6 instances. Despite the marked intermixture of patterns frequently encountered the use of the dissecting microscope revealed a geographic distribution of such nature as to indicate that this occurred only at points of juxtaposition. As one moved away from these zones one or another variant became predominant and relatively discrete. Often one or more patterns recurred in different portions of a tumor producing a mosaic structure. Usually however each type occupied its own segment and was not recurrent within the tumor mass itself, although it was often reproduced in one or more metastatic sites.



Fig 71—Bronchiogenic carcinoma entering the lumen of a major bronchus in the upper margin of the photograph. Ink dots represent the areas of varied histology observed in this lesion.

DISCUSSION

Intraneoplastic pleomorphism is commonly considered evidence of tumor cell multipotentiality. This need not however, invariably be the case. If one visualizes the panoramic distribution of patterns in a given tumor by means of the dissecting microscope multipotentiality does not always appear to be a logical explanation for the existing arrangement. Under these circumstances it would seem more reasonable to attribute the phenomenon to coalescence of

TABLE I PULMONARY CARCINOMA STRUCTURAL PATTERNS

1 type	42 cases
2 types	27 cases
3 types	12 cases
4 types	4 cases
5 types	2 cases

2 or more neighboring neoplasms, each of which arose independently and with different polarization. Thus there would result intermixture of structural types at points of impingement but retention of the original architecture throughout the remainder of the lesion.



Fig 7 B—A juncture zone in Fig 7 A demonstrating adenocarcinoma on the left and squamous cell carcinoma on the right.

Fig 7 C—A region in Fig 7 A showing large acinus mucus secreting adenocarcinoma.

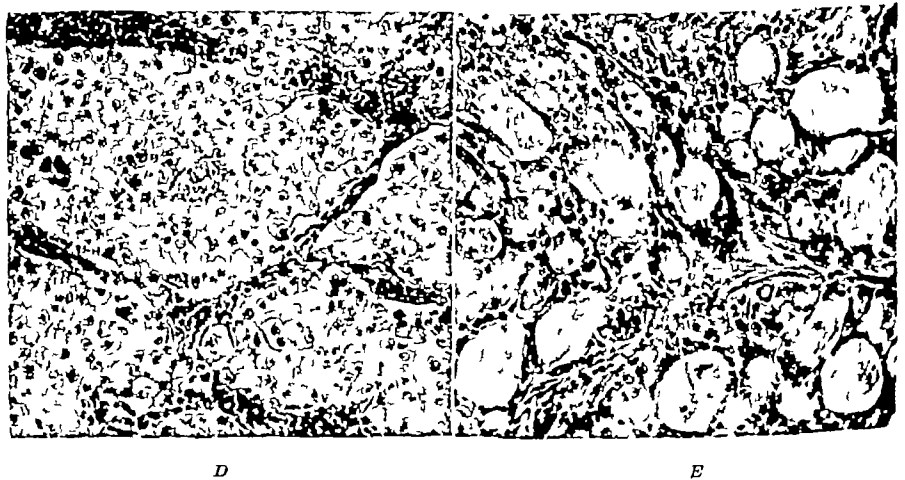


Fig 7 D—Another zone comprised of alveolar squamous cell carcinoma.

Fig 7 E—In this area the tumor has the structure of a small acinus adenocarcinoma. The secretion here is nonmucinous.

Coupling the complexity of histologic patterns with the frequent occurrence of independent and contiguous neoplasms of both gross and microscopic size (Table II), it would seem reasonable to presume that many bronchiogenic carcinomas arise not from one but from several proximate sites. In only 33 of

the 87 cases were none of the features considered contributory to this hypothesis detected. The evidence becomes even more convincing when one appreciates the fact that in many cases in which the pertinent lesions were observed they appeared in only one of several sections while in other tumors in which none were found only single blocks had been sectioned. In the gastric carcinoma study when additional blocks were made in stomachs which had failed to exhibit the lesions searched for they often became manifest in sections prepared in other areas. Thus there is reason to believe that serial sectioning of the lungs also would probably increase the incidence rate.

There exists doubt in some quarters that the process termed preinvasive carcinoma is an irreversible one and inevitably proceeds to the invasive state. Frankly, albeit early, invasion was observed to stem from several *in situ* plaques in this series (Fig. 3B). This by no means settles the problem of inevitability of invasion but certainly substantiates the concept of malignant potentiality.

TABLE II PULMONARY CARCINOMA EVIDENCE FOR MULTIPLE SITES OF ORIGIN

None		35 cases
Multiple gross neoplasms	5	32 cases
Independent foci microscopic neoplasm	19	
Contiguous plaque phenomenon	13	
Varied histologic pattern	46	

In this respect it is important that we clarify the distinction between preinvasive carcinoma and squamous metaplasia of bronchial mucosa. In the examples we have accepted we believe the distinction to be unequivocal (Fig. 4,B). Squamous metaplasia was an exceedingly common occurrence in these specimens and though occasionally exhibiting isolated cell or nuclear atypism, such areas always appeared to retain both polarity and orientation and rarely revealed significant mitotic activity. So far as the data here presented are concerned, when any question existed in this respect the lesion was not classified as neoplastic.

Perhaps the matter of distinction between independent neoplasms and metastasis requires further clarification. Among the small group of grossly recognizable lesions, this distinction could not be made with certainty, although the relation to bronchial passages and the cytologic variation led us to believe that they were indeed independent tumors. The preinvasive plaques, on the other hand, were so characteristic were wholly unrelated to lymphatic extensions and showed so little similarity or architecture to the grossly presenting tumor that there seemed little room for doubt of their independence of origin.

SUMMARY

Large tissue sections were prepared from 87 lungs containing bronchiogenic carcinoma.

In 5 of these there were 2 grossly visible primary carcinomas in the same or different lobes.

In 25 other specimens there were found, on microscopic examination, plaques of preinvasive or early invasive carcinoma both in close contiguity with the main tumor or at such a distance from it as to be considered independent lesions.

In 53 per cent of the cases the major presenting tumor contained 2 to 3 different histologic patterns arranged in such manner as to imply initial independence and ultimate fusion of neighboring neoplasms

The majority (63 per cent) of the specimens examined exhibited one or more of the features enumerated above

It is believed that the aggregate of these observations lends weight to the hypothesis that bronchiogenic carcinoma may often arise from more than one site and that frequently clinical recognition does not occur until coalescence of independent growths has been completed

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DISCUSSION

DR J R. MC DONALD, Rochester, Minn.—I wish to congratulate Dr McGrath on the interesting observations he has reported. I wish to reiterate some of the observations we have made in connection with proximal extension in squamous cell carcinoma of the bronchus. We have noted for some time that between 15 and 20 per cent of squamous cell carcinomas of the bronchus show this noninvasive phenomenon proximally. This is of great importance to the thoracic surgeon because the noninvasive part of the lesion cannot be recognized grossly. Therefore, if the surgeon cuts across it there will be a recurrence in the stump, and I think that accounts for a great many stump recurrences in resections of bronchiogenic carcinoma of the squamous type.

The other thing is that one case has been reported in the literature by Papanicolaou in which the entire squamous lesion in the bronchus was in situ. I think this is important because in this particular case there was a positive cytologic examination, and many physicians and surgeons would have accused the cytologists of issuing a false positive report. As it was, the case was resected and unquestionably the patient was cured.

DR H. BRODIE STEPHENS, San Francisco—I should like to ask Dr McDonald how one can tell from cytology alone on which side the carcinoma is? That refers to the case recently reported by Dr Papanicolaou.

DR J R. MCDONALD, Rochester, Minn.—That is a very difficult problem. Differential cytology on bronchial secretions would help but in differential cytology, of course, there may be a spillover from one bronchus to another. The only positive way would be to obtain a biopsy, with the chance of obtaining a biopsy by bronchoscopy. But I do not think one should blame cytologists for every false positive that occurs.

DR MC GRATH (Closing)—There may be some answer to Dr Stephens' question. In Dr Papanicolaou's case there had been repeated bronchoscopies on that particular patient and repeatedly positive cytologic findings from the same side. I would like to utilize some of the remaining time to show tables summarizing our findings.

(Slide.) These are the several patterns which we found, and (Slide) this shows the summaries of the entire study made.

I am grateful that Dr McDonald drew the obvious inferences from this study. One is, emphasis upon the necessity for a high amputation of the bronchial stump. Furthermore, it is our feeling that we are not in the least enthusiastic except under extremely extenuating circumstances, about lobectomy in the treatment of bronchiogenic carcinoma.

THE USE OF SK-SD (VARIDASE) IN THE MANAGEMENT OF EARLY POSTOPERATIVE PARTIAL PULMONARY RESECTIONS

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THE applicability of SK-SD (Varidase) as a therapeutic measure against fibrinous, purulent, and sanguineous collections has been well demonstrated.¹ Used within the thorax the enzymes have been shown to be practical and of considerable value in a variety of situations arising in medical and surgical thoracic patients, and previous reports describe in detail their use in hemothorax postpneumonic, and chronic empyemata.⁴⁻⁷ In those reports either total resection of a lung had been accomplished or the presence of lung collapsed by a coagulum presented the problems under consideration among the cases cited.

Instances of partial pulmonary resection were not encountered among the reported patients undergoing treatment with the enzymes. However, their use in one case of lobectomy was accomplished by the author in 1949 with a successful result, but a report of two failures after similar procedures with evidence of fistulae occurred subsequent to that.⁸

Since the enzymes uniquely lyse fibrinous deposits and nucleoprotein, which essentially form the framework of the bronchial stump healing processes,⁹ some hesitation has existed in the application of SK-SD to early postoperative partial pulmonary resection cases. It has been pointed out previously that a fistula may be dormant or occult and prevented from becoming manifest by the tamponing effect of fibrin or debris but, with dissolution of the latter, leakage occurs.¹⁰ For these reasons when the enzymes are used after partial pulmonary resections, it is mandatory that drainage tubes are in situ and suction is available so that the immediate, or delayed, escape of air and fluid follows. Prompt re-expansion of the remaining lung tissue can thus be brought about. There is no evidence that the enzymes create fistulae but only that they become apparent when they already exist, the enzymes have no effect on living cells.¹¹

Creech, De Bakey, and Amspacher, in experimental studies, have shown that the use of SK-SD during the early postoperative period following pneumonectomy did not interfere with the healing process of either the bronchial stump or the ligated vessels when the procedure was properly performed.¹ These same authors emphasized the necessity of meticulous pleuralization of the bronchial stump as a guard against fistulae when SK-SD was used. If proper pleuralization were not accomplished, they inferred that bronchial stump healing then depended upon the integrity of the clot formed about the stump. If this clot were dissolved by the fibrin-lysing system, the framework for early healing would be removed and unless an intact bronchial stump were present

a fistula might become manifest. These technical points were considered essential previously when SK-SD was used in patients following pulmonary resection surgery.¹⁰

Many surgeons no doubt have had the experience of observing good drainage from thoracotomy tubes early postoperatively but not infrequently, for some inexplicable reason after a period of 24 to 48 hours' drainage, becomes sluggish and finally ceases when clotting occurs within the tube. Roentgenograms of the chest then reveal evidences of an accumulation within the operated hemithorax and physical findings reflect the evidence of a space-filling coagulum. In these situations no amount of maneuvering or manipulation with the drainage tubes is of avail. One then may attempt aspiration and get nothing or, at the most, a few cubic centimeters of serous fluid. The operator must then be satisfied with incomplete drainage of the thorax and trust that the residual coagulum will be absorbed, and the density seen by roentgenogram will fade spontaneously. If the situation is worse thoughts may be turned to decortication after a reasonable period of time has passed and further possible clearing appears remote. In either event a well-performed technical procedure is marred by a frustrating complication. Large accumulations within the chest often defy prompt attempts at treatment and therefore demand more attention than small collections which frequently vanish even without aspiration.

With the previous experience gained in the use of the enzymes in various types of hemothorax and with the clinical observation in one case of partial pulmonary resection, elaboration upon the application of the enzymes in similar situations, where their use might be indicated, appeared justified. It was reasoned that by controlling leakage of air and fluid with drainage bottles and suction after the instillation of the enzymes the behavior of the remaining lung tissues would resemble that immediately following operation. Under these conditions re-expansion could take place without the presence of excessive amounts of fluid imbued with coagulating tendencies.

Six cases of partial pulmonary resection, in which evidences of coagula existed, treated early postoperatively with the streptococcal enzymes, constitute the substance of this report. The cases included are as follows:

- 1 Right upper lobectomy and resection of the superior segment of the right lower lobes
- 2 Basilar segmental resection left lower lobe
- 3 Right upper lobectomy and enucleation of caseous tuberculous foci from the superior segment of the right lower lobe, and decortication of the right lower and middle lobes
- 4 Apical and posterior segmental resection right upper lobe with decortication right middle and lower lobes
- 5 Apical and posterior segmental resection right upper lobe with enucleation of foci from the anterior segment of the right upper lobe and superior segment of the right lower lobe
- 6 Enucleation of a coccidioidomycotic cavity from the left upper and contiguous left lower lobes

It is to be noted that in addition to partial resection of lung tissue several combined procedures were performed such as enucleation of tuberculous foci and decortication of the remaining lung tissue.

With so few cases, no claim is made for dramatic results having been obtained in the form of complete clearing by roentgenogram and physical findings. However, the progress of the patient was enhanced in most instances and neither ill effects relative to fistulae were incurred nor was wound healing jeopardized.

The diseases encountered at operation, and diagnosed histopathologically, were tuberculosis, three cases, coccidioidal granuloma, two cases, and bronchiectasis, one case. Cultures for pyogenic organisms and acid fast bacilli were reported negative when fluids were submitted for examination. In no cases was there, at the time of treatment, either laboratory or clinical evidence of an infected hemothorax or empyema.

MATERIALS AND METHODS

The streptococcal enzymes (now known as Vardase) used in this study were prepared by methods described by Christensen¹³ and modified by Lederle Laboratories, Inc.* to meet commercial production requirements.

Coagula forming within the postoperative chest are composed of blood clot, fibrinous gel accumulations, and a combination of products of exudation. If infection takes place empyema may be the end result. In the situations under discussion no evidence of infection was present and the substrate requiring dissolution was sanguineous or fibrinous in quality. The streptokinase component in the dual enzyme system was therefore the important factor in management of the problems under consideration. The presence of the streptodornase increment does not detract from the action of streptokinase. As noted previously laboratory control measurements are not necessary for the practical and effective clinical application of streptokinase except in infrequent instances.⁴

Dosage—Details relative to mixing the lyophilized materials and data concerning the dosages employed are outlined in a previous article.⁴ For the present group of cases at least 100,000 units per injection of SK SD were employed. When the larger drainage tube was selected the enzymes were dissolved in at least 30 cc of saline and if the smaller drainage tube was chosen 20 cc of diluent was used. In only one case was the treatment with the enzymes repeated.

SUMMARY OF DATA RELATIVE TO THE TREATMENT OF PATIENTS

Operative Technique—The anesthesia was conducted with an endotracheal tube in place and usually consisted of nitrous oxide, oxygen, and pentothal with succinyl induction. All the operations were performed through the usual posterolateral approach with silk technique. The rib most advantageously situated near the diseased area was removed subperiosteally. For upper lobe

*Lederle Laboratories, Inc., Pearl River, N. Y., supplied the material used in this study.

disease frequently the fourth rib was removed. Individual ligation technique was used on hilar and subhilar structures. The bronchial stump was meticulously and thoroughly pleuralized. In segmental resections no attempt was made to pleuralize the raw surfaces. Before closure a No. 22 French catheter with two holes was introduced through the second anterior interspace and fixed to the cupola of the hemithorax. A large fenestrated latex rubber tube was introduced through a subjacent rib space and this was connected to a water seal during closure. Both tubes were connected to water seals and suction was added during the early postoperative course. Controlled suction was available at the time of treatment with the enzymes. All the patients received penicillin pre- and postoperatively and those with a diagnosis of tuberculosis in addition received streptomycin. One patient also received terramycin.

When a patient was selected for treatment one of the drainage tubes, usually the lower one, was clamped off approximately 2 inches from the chest wall. The length of tube proximal to the clamp was then painted with Merthiolate or with another suitable antiseptic agent. The dissolved enzymes were drawn into a syringe with additional saline and forcibly injected into the chest through an attached 18-gauge needle inserted into the tube through the prepared area. If the smaller upper or anterior tube was used the mixture was instilled simply by attaching the syringe to the lumen of the tube.

Although streptokinase, when instilled immediately, activates the lysing system responsible for the dissolution of fibrin localizations and lysis of coagula within the thorax, the completion of the process occurs at variable periods of time. This time factor is essentially determined by the physical form of the coagulum and the extent of contact between enzymes and substrate^{3,4}. However, a considerable portion of the expected lysis may occur within three to six hours and this treatment time was arbitrarily chosen for the cases observed in this article.

Careful observations were made of the patients treated and, if the clinical picture necessitated termination of the treatment, the treatment time was reduced accordingly. However, this occurred on only one occasion but in another instance at the end of six hours one patient had some respiratory difficulty which was relieved by the opening of his drainage tubes with the accompanying escape of air and fluid. Observations were made for the escape of air when one tube was clamped off, the other remained open and measurements were made of the fluid presumably produced as the result of the action of the enzyme system. In addition to frequent physical examinations, chest roentgenograms were made as soon after treatment as possible and subsequently as indicated.

Untoward manifestations directly due to the use of the enzymes for the most part consisted of rather prompt rise in the temperature, usually about 2° F. The febrile response lasted only about twenty-four hours by which time the fever had reached a peak and dropped precipitously to the original pretreatment level or lower. Minor toxic manifestations consisted of loss of appetite, malaise and a few patients complained of increased pain.

CLINICAL RESULTS AND CASE REPORTS

PATIENT MRS G R—This 43 year old white woman had known tuberculosis since April, 1949. A roentgenogram then revealed cavitary disease in the right upper lobe. Following the discovery of her disease she was hospitalized from May to July, 1949, and a right pneumothorax was attempted. She received 1 Gm. streptomycin twice a week and Paraminosalicic acid during this hospitalization. She was discharged on modified bedrest. She continued her modified bedrest at home for a year and one half when she again began to increase her privileges although the roentgenogram at that time did not show complete healing of the disease in the right upper lung field. In June, 1951, her sputum was re-examined and found to be positive for AFB. A roentgenogram of the chest revealed heterogeneous densities in the right upper lobe region with areas of radiolucency. Her past history was noncontributory except that in January of 1950 she was found to have mild diabetes and required very small doses of insulin on occasions but usually the diabetes was controlled by diet alone.

Physical examination and review of the roentgenograms disclosed evidence of chronic tuberculosis fairly well confined to the right upper lobe region.

Operation July 12, 1951—The apical and posterior segments of the right upper lobe were found to be markedly shrunken, nodular and firm. There was a complete pleural fibrous symphysis over this region and extrapleural dissection was necessary. The anterior segment of the right upper lobe was reduced in size and in addition presented two large nodular areas within its substance. Focal areas of disease were palpable in the superior segment of the right lower lobe. A right upper lobe lobectomy and a superior segmental resection of the right lower lobe was performed. A catheter was introduced anteriorly and fixed in the cupola of the hemithorax and a large fenestrated latex rubber tube was introduced posteriorly for drainage purposes.

Histopathological diagnosis Tuberculosis

Postoperative Course—The patient's temperature rose to 104° F (rectally) by the second postoperative day but thereafter fell by lysis and was low grade by the fifth postoperative day. Both water seal bottles connected to 15 cm of water suction drained fluid and air in moderately large quantities through the second postoperative day. On the third postoperative day there was very little drainage and no further air leaks were noted. By the fifth postoperative day the lower tube had stopped functioning although the upper tube continued to drain small amounts of fluid. The lower drainage tube was removed that day but the roentgenogram taken at that time disclosed evidences of either fluid or a coagulum in the lower lung field. The breath sounds had gradually become more diminished. On the eighth day additional roentgenograms of the chest showed increased amount of fluid in the operated chest and on attempted aspiration only 10 cc of bloody fluid could be aspirated from two posterior sites (Fig 1).

The anterior tube had stopped draining. Ampules of SK SD (Varidase) were injected simultaneously into the anterior tube and into the chest where small amounts of bloody fluid could be obtained. The upper drainage tube was allowed to remain open. On July 21, twenty four hours after the injection of the Varidase, 500 cc of fluid had drained into the bottle and, in addition, 75 cc of fluid was aspirated from a selected posterior site. The following day, July 23 (three days after SK SD), roentgenograms and fluoroscopy revealed very marked clearing of the hemithorax (Fig 2).

The upper tube was then removed and the patient was discharged three days later, on July 25, for further convalescence.

The patient was seen intermittently one month, three months, and eight months following operation. By fluoroscopy and roentgen examination, the operated chest continued to show clearing and her postoperative course was considered entirely satisfactory (Fig 3).

A subsequent report from her physician indicates that the patient has negative sputum and continues to be recovering satisfactorily.

Comment—This patient had resection surgery for tuberculosis and postoperatively presented evidences of incomplete drainage of the operated hemithorax with roentgen evidences

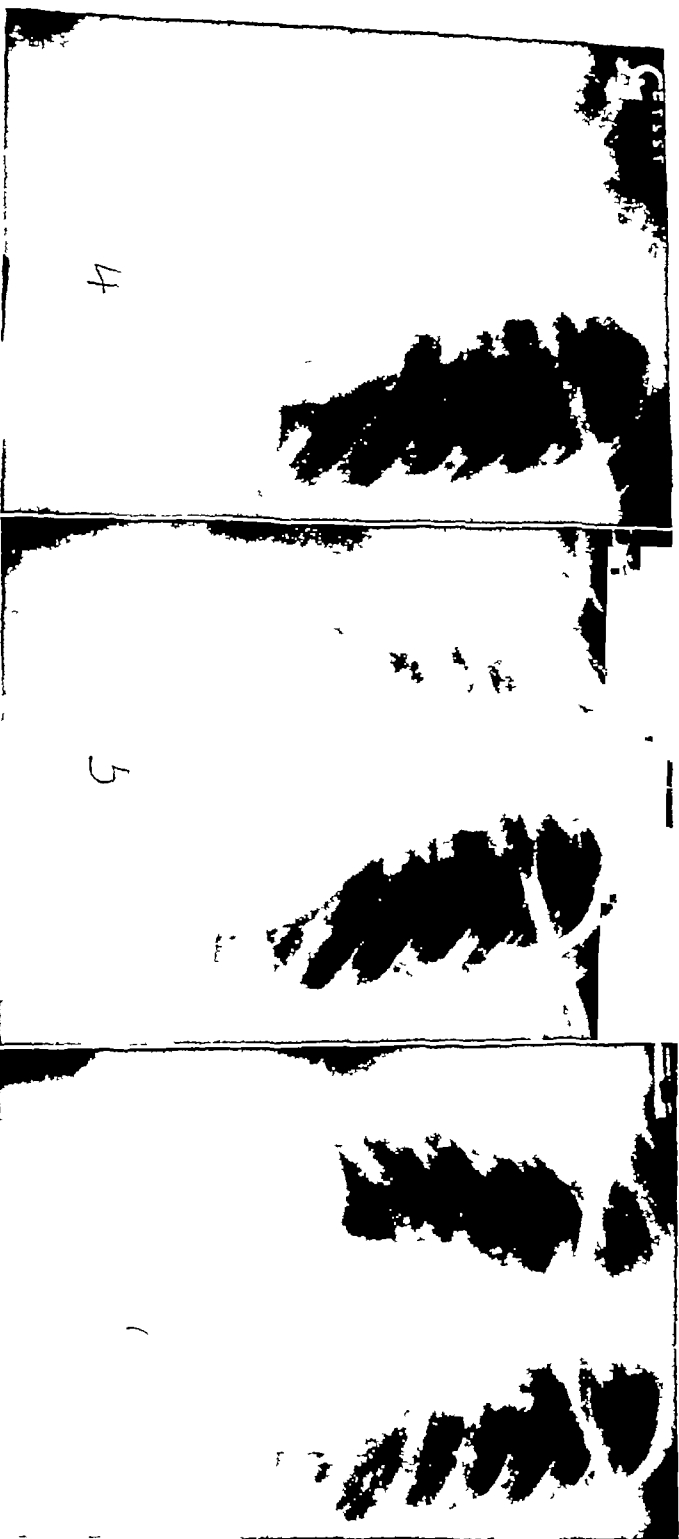


Fig. 1

Fig. 2

Fig. 3

Fig. 1 (Patient Mrs. C. R.)—Roentgenogram appearance of the ribbed hemithorax on July 20, the eighth postoperative day immediately before the insertion of the empysem into the chest (the lower or posterior tube had been removed previously).
 Fig. 2 (Patient Mrs. G. R.)—Roentgenogram of the chest taken July 23, three days after treatment with the empysem. There has been dramatic clearing, and there was comparable improvement in the physical findings over the operated side.
 Fig. 3 (Patient Mrs. G. R.)—Posteroanterior roentgenogram of the chest about five months following operation showing further clearing of the postoperative pleural reaction.

of increased opacity of the operated side and diminished to absent breath sounds on physical examination. Following the introduction of Varidase there was a prompt drainage of the liquefied coagulum with a small amount of leakage of air which permitted complete re-expansion of the remaining portion of the lung and no evidence of chronic fistula was ever encountered. Follow up has been continued for approximately eight months and there is continued clearing of the operated chest without evidence of recrudescence of the disease.

PATIENT O J—This 27 year old white man had left lower lobe pneumonia at the age of five years. He had cough and sputum as long as he could remember and his sputum was frequently purulent and varied in amounts. Intermittent mild hemoptyses occurred for almost ten years.

Essential physical findings were confined to the chest where numerous rhonchi and medium moist râles were heard over the left lower lobe posteriorly. The bronchographic examination disclosed cylindrical bronchiectasis in a shrunken basilar division of the left lower lobe. The remaining portions of both lungs were within normal limits.

Initially the patient brought up over an ounce of purulent sputum daily but with anti-microbial therapy and aerosol penicillin this was reduced to scant amounts of mucoid sputum.

Operation, November 5, 1951—A segmental resection of the basilar segments of the left lower lobe was performed. The chest was drained only with a large posterior tube in this instance and no anterior tube was used.

Postoperative Course—The tube bubbled for about forty eight hours and drained about 500 cc of bloody fluid. Tracheal suction was performed frequently following operation. His temperature rose immediately after operation and reached a maximum of about 103.6° F on the fourth postoperative day. His breathing on the third day was somewhat difficult despite tracheal suction and bronchoscopy. Roentgenographs of the chest showed a marked opacity in the left hemithorax and SK SD was instilled into the posterior tube. Subsequently there was slight clearing with drainage of 200 cc of fluid but two loculated areas appeared anteriorly and superiorly over the left upper lobe. Aspiration was of no benefit. The tube was removed on the fifth postoperative day. By the sixth day it was quite evident that a large coagulum existed over the left upper lobe. Additional taps failed to yield any fluid. The temperature was 103° F from November 11 to November 14.

On November 15, the chest was reopened and decortication was performed. Upper and lower tubes were inserted. On November 16, the day after decortication, additional SK SD was instilled into the upper tube and there was a prompt drainage of about 300 cc of fluid in the lower bottle. On November 19, the tubes were out, but there was some increased density over the lower lung field. The temperature again rose to 103° F and there were a few râles in the upper chest. However on increased medication including terramycin and streptomycin the temperature fell by lysis and was normal during the subsequent forty eight hours. The sutures were removed on November 21 and roentgenograms on November 27 showed very much improvement. All the cultures obtained from the chest were negative. The sputum disclosed an organism resembling *aerobacter aerogenes*. The patient was discharged November 24 for further convalescence and has shown marked clearing in the operated side. He returned to work and has had no further complaints.

Comment—At the time of the second thoracotomy it was noted that the only clean pleural surfaces existed in the region where the posterior tube had functioned. The enlarged superior segment of the left lower lobe showed a small area of induration adjacent to the diaphragm to which it was firmly fixed. The segment was well aerated and could be easily reinflated whereas the upper lobe which was covered with the recently formed peel and fibrinous exudate was difficult to reinflate. It is to be noted that although SK SD was instilled into the lower tube complete liquefaction of the coagulum in the entire hemithorax did not occur. This was thought to be due to the fact that early organization had begun and limited the area of treatment, preventing the enzymes from permeating the collection, anteriorly and superiorly over the upper lobe region. Because at the time of aspiration no definite evidence could be obtained that the needle was in an area of fluid or coagulum, enzymes were not instilled into this region. In previous reports a word of caution was rendered to make sure that the needle was in the proper position and some fluid could be

aspirated before the enzymes were instilled. Observing such precautions prevents the injection of the enzymatic solution into the lung parenchyma. In this case if an interior tube had been used perhaps the foregoing complication could have been circumvented.

An additional point to be gained in this situation, although not classified as an example of successful treatment with the enzyme, is that on the day following decortication enzymes were instilled into the operated area in an effort to hasten the liquefaction of any residual or newly formed coagulum. Despite the short interval (one day) between operation and the use of the enzymes there was no evidence of leakage through the thoracotomy site and the healing of the wound occurred unaltered. The patient was examined approximately five months after operation and has shown almost complete clearing of the operated chest.

PATIENT MRS. H. G.—This 27 year old white woman was first examined on July 17, 1951. In March 1951, the patient was seen by her physician for what was diagnosed as a "strep throat." She had a moderately productive cough and some evidences of sinusitis. She had lost some weight and had malaise. Sputum obtained by the physician was positive for acid fast bacilli. A roentgen examination of the chest disclosed evidences of fibroulcerative tuberculosis of the right upper lobe and a large nodule subjacent to this region thought to be in the superior segment of the lower lobe. In January 1948, when this patient was hospitalized for childbirth roentgenogram of the chest disclosed a similar large density in the right upper lobe region. At that time she had no symptoms and the roentgenographs were interpreted as showing chronic pneumonia.

Positive physical findings were confined to the chest where dullness to percussion over the right upper lobe was apparent. Breath sounds were harsh in this region but no rales were audible. The left side was entirely negative.

Operation August 1, 1951.—Upon entering the right hemithorax a complete pleural symphysis was found. The right upper lobe was found to be shrunken, airless, nodular, and firm. A thin fibrous envelope surrounded the lower and middle lobes when they were freed. The right upper lobe was removed by extra and intrapleural dissection. The major and minor fissures were incomplete and sharp dissection was necessary to remove the upper lobe from the contiguous portion of the adjacent middle and lower lobes. One large firm nodule was palpable in the superior segment of the right lower lobe and this was enucleated. A second smaller nodule inferior to the larger one was likewise enucleated. The areas in which the enucleation was accomplished were approximated with mattress sutures of fine silk. The fibrous peel over the remaining portion of the lung was removed by blunt and sharp dissection. This permitted good re-expansion of the middle and lower lobes on re-inflation. It was noted then that there was poor aeration of the firm superior segment of the lower lobe. The chest was closed in the usual manner and posterior and anterior tubes were inserted for drainage purposes.

Postoperative Course.—During the first postoperative day the breath sounds over the operated side were satisfactory. The usual amount of drainage into the water seal system and bubbling indicating air leakage continued for the first twenty four hours. Late the first postoperative day a leakage in the large drainage tube was discovered where it was attached to the glass tube. This leak was repaired but on examination of the chest the breath sounds were found to be diminished and poor drainage from the hemithorax was suspected. On the second postoperative day, August 6, roentgen examination showed an opacity over the entire operated side (Fig. 4).

Because of the preceding events and the possibility of coagulum having collected within the operated chest, ampules of Varidase were instilled into the upper and lower tube. It was the plan at that time to leave them clamped for a period of from four to six hours. However at the end of three hours the patient complained of some shortness of breath with increased chest pain. It was thought that her complaints were more apparent than real but nevertheless the symptoms were accepted as bona fide and three hours after the Varidase had been instilled the tubes were opened. There was an immediate escape of about 100 c.c. of fluid and a few bubbles of air into each drainage bottle. On August 7, the third postoperative day, another 100 c.c. had drained into the upper tube drainage bottle and another 150 c.c. had accumulated in the lower drainage tube bottle. The upper tube at

that time was removed. On the fourth postoperative day, there had been approximately 30 to 75 cc more drainage into the lower tube drainage bottle and physical findings indicated that the breath sounds were improved and roentgen examination revealed some clearing. The lower tube was removed later that day. After the instillation of the Varidase the temperature promptly rose to 102.8° F but in twenty four hours it had subsided to 100° F and thereafter it continued as low grade to normal. On the sixth postoperative day the breath sounds were good but some haziness on the roentgenograms still existed (Fig 5). An aspiration in the posterior axillary line on the day failed to yield any fluid.

Because of the existing density in the upper portion of the hemithorax, its failure to clear with the treatment outlined and due to the fact that an upper lobe lobectomy had been accomplished, a modified thoracoplasty was performed on the seventeenth postoperative day, August 21. This operation consisted of removal of the entire first rib and segments of ribs two and three. The transverse processes and posterior rib stumps were left intact. The patient made an uneventful recovery from the thoracoplasty and was discharged on August 28, one week following the thoracoplasty and twenty four days following the lobectomy.

The pathologic specimen showed fibrocaseous tuberculosis and the nodules submitted for examination showed caseous centers.

At follow up one month and three months after surgery she had made an apparently uneventful recovery. Sputum by culture was negative for acid fast bacilli. Roentgenogram continued to show clearing in the operated chest (Fig 6). The last follow up on this patient was in March, 1952, almost eight months after operation, at which time there was evidence of continued improvement.

Comment—In this particular situation it was thought that due to the leakage of the drainage system a coagulum had been permitted to form and Varidase was used to liquefy this coagulum and permit further drainage. The persistent haziness over the lung was thought in part to be due to the pleural reaction since this patient had decortication of the envelope of fibrous tissue described at operation. No evidence of fistula became manifest and satisfactory reexpansion occurred and progressive clearing by x ray examination was evident.

PATIENT MRS O C—This 34 year old white woman had known tuberculosis since 1941. Following a brisk hemoptysis her sputum was found to be positive. A roentgenogram showed a cavitory lesion in the right upper lobe area. She was hospitalized in December, 1941, and a right pneumothorax was instituted. She remained in a sanatorium for approximately one year and the pneumothorax on the right was maintained. About a year and one half later she was told that her disease was healed and the sputum was negative. Following a pregnancy and a hysterectomy in November, 1943, her sputum was again found to be positive. Hemoptysis recurred in January, 1944, and she reentered a sanatorium. A right phrenic crush was performed in 1945. After continued bedrest, the sputums were said to be negative for acid fast bacilli on culture and the disease was thought to be arrested. She gained about twenty pounds and was working as a student x ray technician when she was reexamined in June, 1951, following some streaking, and the sputum was again found to be positive. At the time of the examination in June, 1951, she had minimal symptoms and scant cough and sputum.

Physical examination yielded contributory findings only in the thorax. On auscultation over the posterior right upper lobe region the breath sounds were of increased intensity. No adventitious breath sounds were heard. The left side was completely negative. At fluoroscopic examination the right diaphragm was seen to move sluggishly. On roentgen examination, heterogeneous densities were present in the right upper lobe region posteriorly. Tomography disclosed evidences of a cavity in the apex of the right upper lobe.

Operation July 20 1951—On opening the right hemithorax a total and complete fibrous pleural symphysis was found to exist. When the lung was freed it was noted that a fairly thick fibrous envelope enclosed it. The pleural peel was subsequently stripped from the remaining portions of the lung following the resection of the apical and posterior segment. A few small nodules were found in the extreme posterior part of the anterior segment and

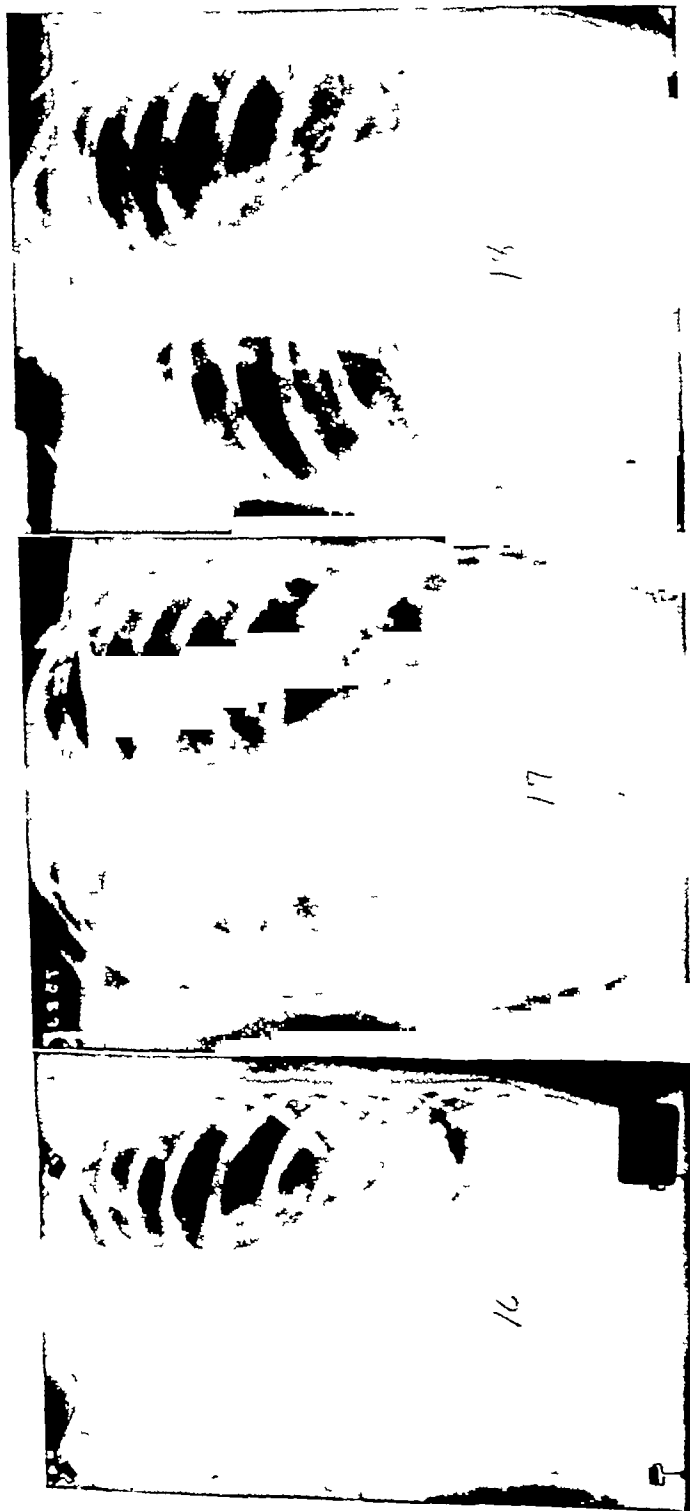


Fig. 1

Fig. 5

Fig. 6

Fig. 4 (Patient Mrs. H. G.) — Depicts the roentgenogram appearance (first postoperative day) before treatment with SK-SD (Varidase). A large collection obscures the entire right hemithorax except a small portion of lung at the base.

Fig. 5 (Patient Mrs. H. G.) — The roentgenogram taken on the tenth postoperative day about one week after treatment. There has been moderately good clearing of the density seen previously but the superior portion remains obscured.

Fig. 6 (Patient Mrs. H. G.) — A follow up roentgenogram of the chest made approximately five months after operation. A thoracoplasty has been done and excellent clearing of the remaining lung is evident.

these were removed with the resected segments. The lung re expanded adequately and no collapse of the chest wall was thought to be necessary. The hemithorax was closed in the usual manner and anterior and posterior drainage tubes were inserted.

Histopathological diagnosis Tuberculosis

Postoperative Course—The upper drainage tube bubbled less than twenty four hours. The lower tube no longer bubbled air after thirty six hours and there was no apparent drainage from either tube thereafter. On roentgen examination increasing opacity of the chest was observed and the breath sounds became markedly diminished. On the third postoperative day, July 23, it was thought that a coagulum existed within the chest with clotting in the tubes and further drainage was not likely. Therefore an ampule of Varidase was instilled into the lower tube which was clamped off. The upper tube was allowed to remain open. Four hours later there was 250 cc of drainage from the upper tube. The lower tube was then opened and 200 cc of bloody fluid drained into the bottle. Roentgen examination on the fourth postoperative day, one day following the instillation of the enzymes, showed only partial clearing but there was very marked improvement on physical examination (Fig 1).

Breath sounds came through adequately at this time. The patient's temperature before the instillation of the SK SD was 100.4° F. Within eight hours after injection her temperature rose to 103.2° F and fell by lysis to 101° F the next day and it became low grade within forty eight hours. Because of no further drainage from the upper tube it was removed on the fourth postoperative day, one day following the instillation of the enzymes. On the fifth postoperative day there was only a minimal amount of additional drainage from the lower tube and it was removed. The patient's course thereafter was uneventful. The breath sounds continued to improve and her general condition improved comparably. She was discharged on July 31, eleven days following surgery.

A follow up roentgenogram on the fifteenth postoperative day, August 4, 1951, disclosed marked clearing of the operated hemithorax (Fig 8). Examinations at the County Hospital, and subsequently when the patient returned home on a modified bedrest program, indicated that continued improvement existed. Follow up examination by roentgenogram showed increased clearing (Fig 9). Sputums were negative on culture. The patient was last examined in March, 1952, and continued progress was evident.

Comment—This patient exhibited a marked opacity of the operated chest with absent breath sounds throughout the operated area. The drainage was thought to be inadequate and because of the lack of breath sounds and the opacity in the operated chest seen by roentgen examination it was deemed evident that a coagulum had collected within the operated side. The immediate clearing as indicated by roentgenogram was not striking but subsequent roentgenograms showed very marked clearing and physical findings improved immediately following the use of the enzymes.

PATIENT E C—This 34 year old white man was admitted to the hospital July 31, 1951. His history revealed that in November, 1943, while in the Armed Forces a chest roentgenogram revealed a small area of fibrosis in the right apex. This was believed to be of no clinical significance, and he was returned to full military duty. A follow up roentgenogram in July, 1945, was considered negative. In April, 1949, a roentgenogram taken at a Veterans Hospital showed an infiltration in the right upper lobe. Sputums were negative for acid fast bacilli and he was discharged with a diagnosis of arrested tuberculosis. A subsequent examination in July of 1950, revealed a possible cavity in the right apex with surrounding infiltration. Concentrates of sputum were negative for acid fast bacilli. Repeat roentgenograms including tomographs in December, 1950, revealed a cavity in the right upper lobe. Tuberculin skin tests were positive in the second strength. An equivocally positive Coccidioidin test was present. In May, 1951, he was hospitalized at a Veterans Hospital and culture of the sputum for acid fast bacilli was said to have been positive. This examination, however, was never confirmed. All other sputums on concentrates and by culture were negative for acid fast bacilli.

Physical examination was essentially negative. Differential diagnosis resolved itself about coccidioidomycosis or pulmonary tuberculosis and pulmonary resection was advised.

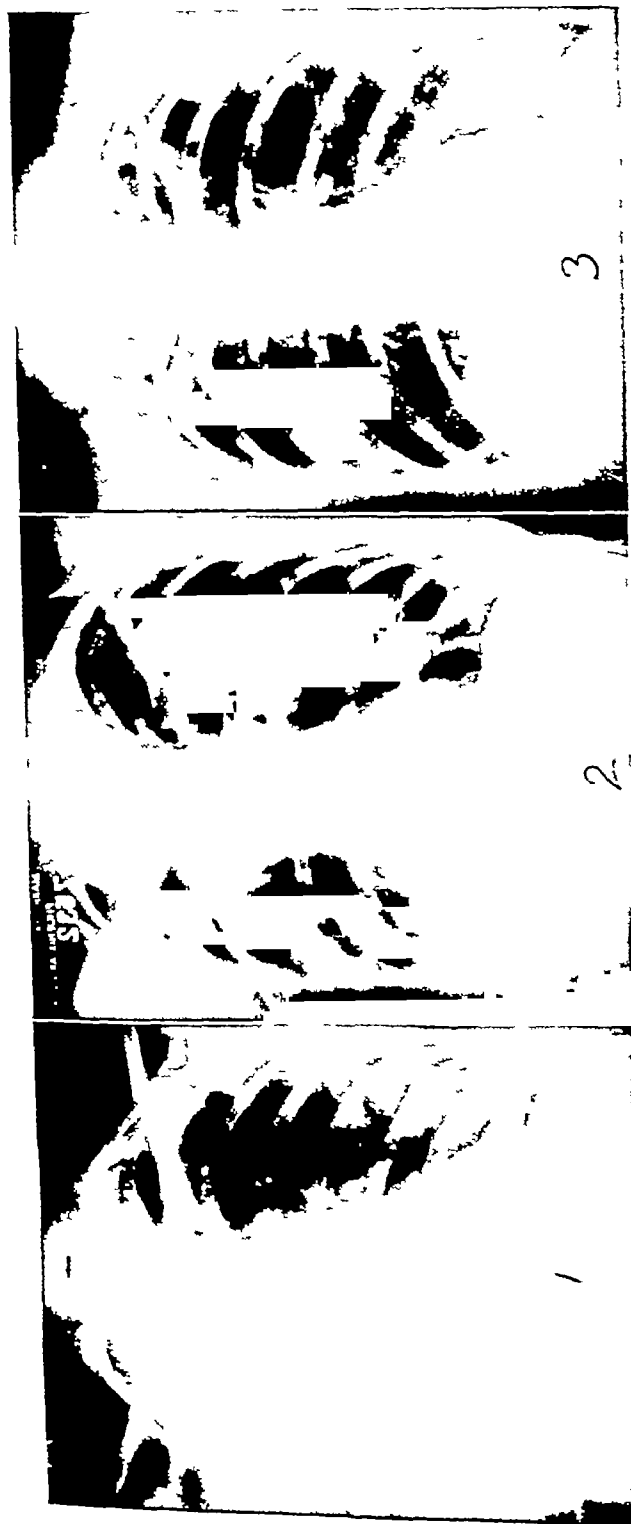


Fig. 7

Fig. 7 (Patient Mrs. O. C.) — This postoperative roentgenogram of the chest was made the day after treatment with the enzyme (Varidase) which was carried out on the third postoperative day. Although there is marked clearing of the operated chest extreme improvement in the physical findings had occurred. A decortication was also performed in this instance and much of the density was therefore considered as pleural reaction.

Fig. 8 (Patient Mrs. O. C.) — A roentgenogram taken fifteen days after reaction and only twelve days following SK-SD therapy. There is noticeable improvement over the previous X-ray and clinical progress was comparable in degree.

Fig. 9 (Patient Mrs. O. C.) — Follow up roentgenogram of the chest made Nov. 4, 1951 approximately three months postoperatively. Further clearing is noted and the patient was clinically well.

Fig. 8

Fig. 9

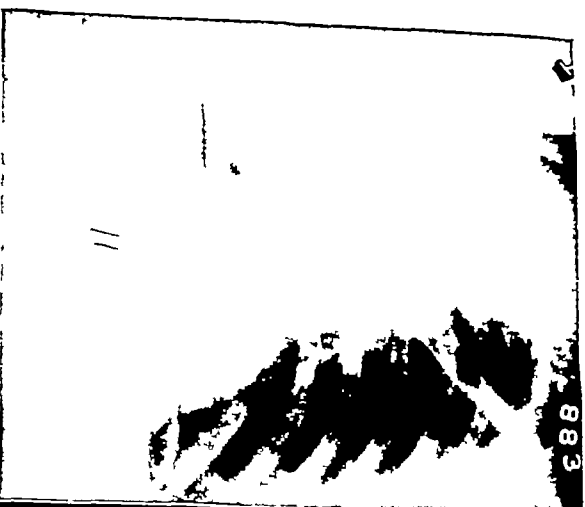
Operation, August 22, 1951—On entrance into the thorax, the right upper lobe was found to be about half its normal size. This was due to the marked reduction of the volume of the apical and posterior segments which were firm and airless. Due to the dense fibrous adhesions extrapleural dissection of the involved segments was required. Two firm rounded foci were palpable, one in the anterior segment of the upper lobe, and one in the superior segment of the lower lobe. These were enucleated during the procedure. The nodule in the superior segment was found to show evidence of central excavation but the nodule in the anterior segment of the upper lobe was solid and both contained amorphous caseous like material. The cavity in the apical and posterior segments was inadvertently entered and showed granular pinkish white walls. There was some collection of detritus covering the lining. The segments were removed in the usual manner and no major leaks followed the resection and the enucleation of the foci. The raw edges of the enucleated areas were approximated with interrupted sutures of fine silk. Before closure of the hemithorax anterior and posterior drainage tubes were inserted.

Histopathological diagnosis of the removed specimen was coccidioidal granulomata of the lung with cavitation.

Postoperative Course—Preoperative laboratory studies indicated the patient's prothrombin time was slightly prolonged, to which too much significance was not attached because other tests, including bleeding and coagulation time, were normal. The prothrombin time was nineteen seconds and the control fifteen seconds. He received 70 mg of vitamin K intra muscularly daily from August 15 to August 21.

Postoperatively there was slow bleeding into the chest cavity, necessitating a total of six transfusions. The drainage tubes by the second postoperative day failed to drain and a large opacity was seen in the upper lung field (Fig 10). His hematocrit the first postoperative day was 41 and he received a pint of blood. The next day the hematocrit was 31 and he received five pints of blood. Because of the accumulated clot in the upper right hemithorax and the thought that bleeding might be due to a prolonged prothrombin time the thought of exploration was discarded and instead treatment with Varidase was decided upon. On the third, the fourth, and the sixth postoperative days ampules of Varidase were instilled into each of the drainage tubes. Each time the enzymes were allowed to remain in situ for four to six hours. On August 25, following an initial instillation, 700 cc of fluid drained within a twelve hour period subsequent to the reopening of the tubes. A total of approximately 2000 cc of bloody fluid escaped into the drainage bottles from the use of the enzymes. Following each instillation there was a leakage of air for a short period of time. After all the accumulated fluid was drained the leakage usually stopped within about six hours. All leakage ceased following the last instillation. The drainage tubes were removed on the ninth postoperative day. Following removal of the tubes there was rapid and continued improvement by physical and x ray examination (Figs 11 and 12). The amount of air leaking into the pleural space while the tube was clamped off was not enough to cause embarrassment of respiration on the first two occasions but the third instillation on August 28 produced some dyspnea after six hours which was promptly relieved when the tubes were open and suction applied. He was discharged from the hospital and was seen three months later at which time no complicating factors were evident.

Comment—In this particular instance it was thought that the bleeding was related to the prolonged prothrombin time. In a previous experience gained by the author following a sympathectomy at which blood loss was unusual in amounts, a prolonged prothrombin time was found to exist. Despite the presence of such a laboratory finding the enzymes were used in that particular instance without any cause for alarm. The enzymes had the desired effect and no evidence of continued bleeding was observed. Re exploration is sometimes an unsatisfactory procedure where the postoperative bleeding is moderate and when performed the source of bleeding frequently cannot be discovered. With these thoughts in mind the treatment in this particular complication was thought best to be as conservative as possible. The enzymes (Varidase) were therefore used. It is to be noted that leakage of air was manifest following each instillation of the enzymes, which was probably the result of dissolution of



Pl. 10



Pl. 11



Pl. 12

Pl. 10 (Patient L. C.) —Roentgenogram appearance of the chest on the second postoperative day depicting the large coagulum in the upper third fourth of the right hemithorax. The tubes at this time were not functioning, and the hemithorax had fallen indicating the likelihood of a blood clot in the operated side.

Pl. 11 (Patient L. C.) —The roentgenogram taken of the chest on the seventh postoperative day following therapy on the third fourth and sixth days and before the tubes were removed. There has been marked resolution of the density seen previously with drainage of a total of 2,000 c.c. of fluid.

Pl. 12 (Patient L. C.) —Post roentgenogram taken approximately one month after emergency treatment. Additional clearing is shown.

the fibrin covering the intersegmental surfaces, but with adequate suction and drainage re expansion occurred promptly in each instance. After the last instillation complete re expansion was apparent and the tubes were removed in the usual manner.

PATIENT MR. R. D.—This 18 year old white man was admitted to the hospital August 1, 1951. His main complaint at that time was cough productive of mucopurulent sputum of several months' duration. In addition he had numerous psychosomatic disturbances and complaints related to various systems. However, all the latter were ruled as having no organic basis and his pulmonary trouble was worked out completely. He had had a cough for about two years but, during the year prior to his admission, he began to raise mucopurulent sputum and occasionally this was greenish or grayish brown. He denied any frank hemoptysis. Work up at the hospital showed a positive tuberculin skin test (first strength) and a positive skin test (1:100) for coccidioidomycosis.

Physical examination was essentially negative except for the chest. A few rhonchi were heard on deep and forced respiration over the left lower lobe posteriorly and in the axillary region. The sedimentation rate was 31 (Westergren method). Sputum was negative for acid fast bacilli on concentrate and by culture. A roentgenogram of the chest revealed a thin walled cavity in the left lower lung field with minimal adjacent mottled infiltrates. This was considered to be a cavity due to coccidioidomycosis and from the history and observation it was thought to be infected.

Operation, Oct. 5, 1951.—On opening the thorax a few filmy and fibrous avascular adhesions were found over the fissure about the midaxillary region. Crossing the obliterated major fissure in its central portion there was a slightly indurated area measuring about 4 cm. in diameter which, by palpation, indicated an underlying cavity. This lesion was found to lie about half in each lobe along the fissure region. During enucleation of the cavity by blunt and sharp dissection from the lobes it was inadvertently entered and a small amount of grayish tan detritus escaped into the wound. Later the cavity was further opened and used as a guide in dissection as one would do in dissecting out a hernia. Very little air leakage and bleeding occurred along the plane of dissection. No attempt was made to close the defect in either lobe. Following removal of the cavity re-inflation of the lung was prompt and only minor air leaks occurred. Before closure a large fenestrated latex rubber tube was introduced through a subjacent rib space and a No. 20 French catheter was introduced through the second anterior interspace.

Histopathological diagnosis. Coccidioidomycosis (by culture).

Postoperative Course.—Following operation there was a gradual diminution of breath sounds over the base of the operated hemithorax. The breath sounds over the upper and anterior portions of the lung were good and on the fourth postoperative day the upper tube was removed but there was evidence of effusion at the base. On the sixth postoperative day mild subcutaneous emphysema was noted in the upper anterior chest and roentgenograms and fluoroscopy disclosed partial collapse of the lung and loculated areas of fluid (Fig. 13). A closed thoracotomy was performed at the second anterior interspace and in addition a thoracentesis was accomplished in the lower anterior hemithorax with removal of about 250 cc. fluid and about 200 cc. of air. Varidase was then injected into the lower tube which remained in situ. On manipulating the tube some of the fluid into which the enzymes had been mixed leaked onto the skin. It was then deduced that the lowermost fenestration in the indwelling drainage tube was near the skin surface. The tube was then fixed tightly within the chest by means of adhesive dressings. The tube remained clamped off for a period of six hours. The upper tube which had been inserted was allowed to remain connected to a water seal and suction. Six hours after the injection of enzymes on opening the tube about 150 cc. of fluid drained into the bottle. Only minimal drainage occurred from the upper tube but air was stated to have escaped. The following day re-expansion was apparent and marked improvement had occurred clinically (Fig. 14). Tracheal suction was instituted and a moderate amount of thick tenacious brown secretions were obtained. The lower tube was removed on the following, the eighth postoperative, day. The upper catheter inserted at

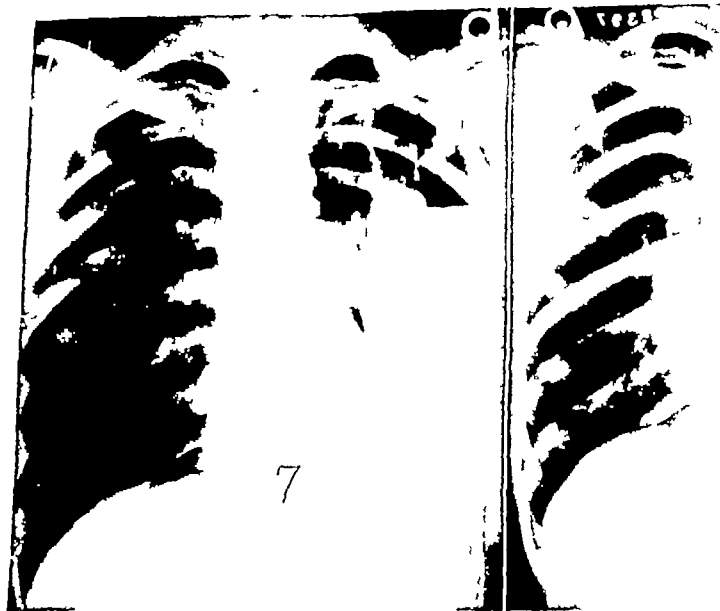


Fig. 13



Fig. 14



Fig. 15

Fig. 13 (Patient R. D.) —Postoperative roentgenogram of the chest taken after discovery of tissue emphysema six days after enucleation of a coccolidial cavity. Hydro-pneumothorax is well demonstrated.

Fig. 14 (Patient R. D.) —Roentgenographic appearance of the thorax the day after the instillation of the enzyme and closed thoracotomy. There is complete re-expansion of the lung.

Fig. 15 (Patient R. D.) —The roentgenogram of the chest made ten days after treatment with Varidase and seventeen days after thoracotomy. A satisfactory result has been obtained with only moderate reaction in the operated area remaining.

the time of closed thoracotomy was removed in three days. The patient showed marked improvement in aeration and clearing by roentgenogram. Subsequently, on the twelfth postoperative day, he was discharged for further convalescence at the County Hospital.

Comment—Enzymes were used in this particular situation in order to dissolve the collected material in the base of the operated chest. It was thought that dissolution of the coagulum would enhance re-expansion which was mandatory. In this instance there was only minimal amount of drainage following the use of the enzyme but there was very prompt clearing both by physical examination and roentgen study. Despite the fact that a large lesion had been enucleated leaving bare surfaces in both the upper and lower lobes, fistulae did not form. It was reasoned that the pneumothorax, seen by roentgen examination, occurred through the improperly placed drainage tube and not through a spontaneous fistula in the operated site, for following the closed thoracotomy and the use of the enzymes, only small amounts of air leakage was evident. The patient was seen at subsequent follow ups as late as March, 1952, and has shown continued improvement although a roentgenogram shows peculiar ringlike pleural markings about the operated area, thought to be of no significance as determined by a bronchographic study (Fig 15).

DISCUSSION

After partial pulmonary resection the healing and restorative processes depend upon the obliteration of air leaks, the natural clotting of oozing surfaces and small vessels, prompt re-expansion of remaining lung tissue, and the elimination of air and exudative fluid from the thorax. Since SK-SD (Varidase) lyses fibrin, its use might be considered a detriment to these healing factors. However, the enzyme system apparently does not prolong bleeding from exposed surfaces or interfere with the clotting mechanism within the lumen of the small vessels as judged by the results in its early postoperative use.

With an adequate system draining the thorax, air leakage from raw surfaces, and the disposal of fluids usually is readily accomplished. Under certain circumstances, however, clotting or gel formation of the drainage occurs faster than it can be delivered from the chest and any damming back of further drainage hastens the formation of a space occupying coagulum about the expanding lung.

Frequently on removal of a drainage tube, a fibrin plug is observed lying within its lumen which no doubt interferes with adequate dispersal of the leakage. By dissolving such a plug and with dissolution of the collection within the chest cavity, on using the enzymes, re-expansion of the lung can be facilitated. Following re-expansion any subsequent drainage is readily discharged before the tubes are removed.

At operation, in three instances, the pleural cavities were exposed to active tuberculous foci during the procedures, yet in no case was there evidence of the infection being seeded or spread in the lung or pleura after the enzymes were used. These patients received streptomycin but several had had the drug previously and no index of the organism's sensitivity was available at the time of their treatment.

In patient O. J., who was reoperated on the tenth day after resection, the results of the previous treatment with the enzymes were observed. In the area where the substances were effective the pleural surfaces were relatively smooth and pliable, although injected, and this region of lung expanded readily on reinflation. The operated surface was firmly fixed to the adhering diaphragm.

and apparently the adhesions remained intact during treatment. There was no leakage from the thoracotomy incision when the enzymes were again instilled the day after the second operation and the wound healed per primam.

The use of the enzymes in patient E. C. was thought to have possibly circumvented a second operation for decortication. As judged by the amount of fluid obtained on treatment and the large density in the involved area on the roentgenogram a sizeable coagulum was present. Despite the prolonged prothrombin time and evidence of bleeding into the area, treatment with the enzymes brought about a favorable result. If the enzymes had adversely affected the clotting mechanisms at the bleeding source there seemingly would have been indications of increased hemorrhage and not termination of it as occurred.

It appears that clotting within the operated thorax and the formation of a fibrin plug in the lumen of the drainage tubes occur not infrequently. One cause for prompt clotting with resulting dysfunction of the drainage system may be attributed to the accelerated coagulation of blood produced by the use of antimicrobial agents.¹⁴

Since the healing processes are not patently slowed and there is no evidence of the enzymes affecting the underlying disease process it would appear that these preparations can be employed advantageously early postoperatively after partial pulmonary resection.

SUMMARY

1 Six patients were treated with SK-SD (Varidase) early postoperatively, following partial pulmonary resection.

2 The procedures were performed singly or in combination and included lobectomy, segmental resection, decortication, and enucleation of disease foci.

3 The diseases encountered were tuberculosis (three cases), coccidioidomycosis (two cases), and bronchiectasis (one case).

4 The enzymes were instilled through drainage tubes already in situ on the second postoperative day (one case), the third postoperative day (two cases), the fourth day (one case), the sixth day (one case), and the eighth day (one case).

5 One patient was reoperated, after segmental resection had been performed ten days previously and the enzymes were instilled the next day.

6 In no instance was there a chronic fistula created or evidence of deleterious effect on the underlying disease process.

7 All the wounds healed per primam and no leakage was manifest into or from the thoracotomy incisions.

8 The results were satisfactory clinically and favorable from a roentgenologic viewpoint.

ADDENDUM

Since the data for these cases were compiled, three additional cases in which the enzymes were used early postoperatively following pulmonary resection procedures have been treated successfully with similar favorable results.

1 A patient following decortication of the left lung and enucleation of multiple foci from the left upper lobe received the enzymes on about the tenth postoperative day.

2 A patient who had a right middle and right lower bilobectomy was treated on the fourth postoperative day

3 A patient who underwent an exploratory thoracotomy with a preoperative diagnosis of carcinoma was found to have a granulomatous lesion in the right lung from which generous biopsy material was removed. This patient had an injection of the enzymes on the sixth postoperative day

None of these patients had any deleterious effects in the use of the enzymes and a prompt and satisfactory postoperative recovery

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DISCUSSION

DR C F DANIELS, New York—We are indebted to Dr Read for his studies presented today and two years ago which have contributed much to the management of postresection hemothorax. If a small rent is made in the pericardium at the time of resection and is followed by hemothorax, the development of cardiac tamponade may follow the administration of streptokinase and streptodornase. Such a situation developed in a patient who had a middle and lower lobectomy, parietal pleurectomy, and partial decortication of the upper lobe for what was believed to be a mediastinal tumor eroding the intermediate bronchus and adherent to the pericardium. A small rent in the pericardium was left uncovered. Postoperatively, clotted hemothorax occurred. On the eleventh postoperative day intrapleural administration of streptokinase and streptodornase was followed by drainage of 900 c.c. of serosanguineous fluid. The next day it was evident that serious cardiac tamponade had occurred. Apparently with partial lysis of the clot occluding the small pericardial window, fluid flowed into the pericardium but a ball valve mechanism prevented its egress. The emergency aspiration of 300 c.c. of serosanguineous fluid gave immediate relief of symptoms. Further intrapleural administration of the enzymes resulted in clearing of the hemothorax without recurrence of cardiac tamponade.

DR MICHAEL DE BAKEY, Houston, Texas—Dr Read, I think, has provided convincing evidence of the value and safety of these agents in the immediate postoperative period under these circumstances where it is desirable to remove pleural effusions that accumulate and prevent prompt expansion of the lung and obliteration of the space. He referred to some experimental studies that we did about a year ago, with the purpose of determining whether or not there was any jeopardy to the healing process following pulmonary resection if these agents were used in the very early postoperative period. It was obvious from these studies that the healing process was not jeopardized if the pulmonary resection was properly performed and, on that basis, we used these agents clinically in six cases, three patients following pulmonary resection under circumstances similar to those described by Dr Read, and in three cases of esophageal resection. In no instance was there jeopardous healing following their use, and the advantage which resulted from their use was similar to that described by Dr Read. We believe, therefore, that our experimental as well as clinical observations can add confirmation to those which Dr Read has presented.

DR READ (Closing)—I want to thank the discussors, especially Dr De Bakey. I was privileged to use his paper before it was published, he was kind enough to send me a copy for use in preparation of this manuscript.

I want to emphasize the very great importance of having tubes in situ when this material is used under the related circumstances. If this is not done you can almost guarantee that something catastrophic is going to occur. I would strongly advise also to have suction available, and also the possibility of even introducing another tube if it becomes necessary. There is one case which I was not able to report because of the time allotted. That is an incident, that will be included in the published manuscript, of a young man in whom I did a basal segmental resection of the left lower lobe. Postoperatively, drainage was very sluggish. I might add that I did not use an upper tube, in other words, it was only single tube drainage. During my training, several men impressed upon me the importance of using two tubes. However, in this instance only one tube was used. It seemed to be an easy situation and I thought that maybe the postoperative course might be hastened. Rather early in the postoperative course it was obvious that drainage was going to be difficult. Coagulation occurred, and the enzymes were instilled through the lower tube. However, they were instilled about the fifth day. Previously we reported that organization of coagulant can occur as early as the fifth day. There was a large pocket superiorly in this case and it was my assumption that the enzymes never permeated this large coagulant. There was evidence of results in the lower portion where the enzymes did reach and infuse about the coagulant. Additional aspiration superiorly yielded no fluid. We have emphasized the importance of obtaining fluid before one instills the enzyme material. None could be obtained in this instance, therefore enzymes were not instilled superiorly.

Subsequently, on about the tenth postoperative day, this man was reopened and a large coagulant and fibrous gel overlying the entire lung had to be decorticated and removed. At that time we made an observation of the segmental resection and the effect of the treatment of the enzymes in this area. As we had strongly suspected but never observed previously, the remaining superior segment, which was quite large in this case, was quite smooth and pliable, the pleura was injected, and it was firmly adherent to the diaphragm along the segmental surface. It was pleasing to note that the area where the enzymes had been effective seemed to show a very good response to the treatment.

REHABILITATION IN THORACIC SURGERY

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TO DATE, relatively little attention has been given to the rehabilitative phase of the postoperative care of patients who have undergone thoracic surgery. The Veterans Administration has an active program in this vital field, but, generally, comprehensive programs do not exist. As a rule, tuberculosis sanatoria adhere to the old method of encouraging their postoperative cases to spend many hours lying supine on a supporting bolster, the objective being to "stretch the spine." Obviously, this procedure does nothing to combat the muscular atrophy and atony, the fibrous matting of the affected soft tissues, the contractures and the potential deforming scoliosis. On the contrary, it actually encourages all these undesirable pathologic changes frequently leading to progressively increasing lateral shifting and rotation of the spinal column and the ribs. The postoperative atrophy and dysfunction of the important muscles of the affected side are no match for the tonic muscles of the opposite unaffected side leading to imbalance of the muscular and ligamentous relationship with consequent distortion.

Starting with this basic concept of the residual postoperative pathology our problem was to

- 1 Find effective means of preventing atrophy of the affected muscles and lessen fibrosis and matting of the soft tissues of the involved side, since the resultant adhesions may seriously hinder restoration of function

- 2 Anticipate and thus prevent compensatory contractures of the muscles of the unaffected side, thereby limiting the apparently inevitable scoliotic torsion of the unbalanced spine. Obviously, the ultimate, complete restoration to normal cannot always be attained, but much can be done to lessen the ultimate disability and functional handicap

- 3 Combat fibrotic and arthritic changes in temporarily immobilized joints, a complication which, if ignored, may well result in permanent "freezing"

Naturally, the more extensive the operative procedure, the more severe is the pathologic residuum, its anatomic distortion, and its physiologic malfunction. A "seven-rib" thoracoplasty resecting ribs, transverse processes, ligaments, muscles, etc., leaves profound anatomic and functional devastation. Not the least of the latter is the extent of damage to the nerve and blood supply of the sectioned muscles frequently irreversible. The more serious the nerve damage the less satisfactory will be the rehabilitation of the affected muscles.

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Aside from the immediate sequelae of the surgical procedure, it is necessary to anticipate the potentially disabling effect of prolonged bed rest so essential in the treatment of the tuberculous patient. This is particularly noxious in cases wherein the patient assumes a comfortable but relatively distorting position while at rest. While this problem is primarily met with in the rehabilitative care of the tuberculous patient, the problem is similar in all post-thoracic surgical care regardless of the etiology.

Extensive surgical procedures require the division of

- 1 The scalenus group of muscles which are detached from their insertion on the first and second ribs. These tendons are not reattached.

- 2 The corresponding parts of the trapezius, rhomboids, serratus magnus, latissimus dorsi, and the pectoralis minor, all of which are reapproximated and sutured.

Since in thoracic surgery, particularly in tuberculous cases, the operative procedures are customarily carried out in "stages," the resultant surgical trauma and all its sequelae require repetitious therapy two or three times in each case. No sooner do the sectioned muscles begin to heal than a new operative stage separates the sectioned tabs.

It should be reiterated that all sectioned muscles other than the scalenus group are reapproximated and sutured. The healing process progresses slowly. Therefore, during the early postoperative stage, rehabilitative exercises must be given with extreme caution and patience.

It should be emphasized that in thoracoplasties, the bony support of the sectioned muscles is gone, and the weakened muscles now constitute an integral part of the thoracic wall and must bear their share of the potential strain and stress. Therefore, one of the ultimate aims must be to strengthen these muscles to the very maximum of their potential.

Furthermore, the absence of the bracing ribs results in a positional shortening of the rhomboids, the trapezius, etc. Studies in the physiology of muscular activity have demonstrated that a muscle functions best when the contraction is initiated with the muscle in its normal resting length. Shortening, therefore, leads to inefficient function. In addition, atonicity of the muscles supporting the corresponding scapula, may, in some instances, lead to a "dropping" or a sagging of the scapula causing concomitant dysfunction and often irritation of the underlying area. The patient, in an effort to avoid the incidental discomfort, voluntarily "locks" the shoulder in an elevated position generally referred to as a "high shoulder" with its compensatory "high pelvis" on the same side. This malposition often leads to contraction of the trapezius and limitation of function of the shoulder girdle. It is not unusual for the surgeon to attempt to solve this problem by removing part of the irritating lower rib or with a partial capsulectomy. Adequate therapeutic exercises can usually prevent this "dropping" of the scapula, obviating such secondary surgical procedures. As noted previously, another complication of thoracic surgery is the fibrosis and ankylo-

sis of the corresponding shoulder girdle. In an effort to lessen postoperative pain and discomfort, the patient deliberately splints the involved area by holding the arm and shoulder girdle rigidly at the side. Unless this is anticipated and prevented by proper posturing and active and passive exercise, permanent severe "freezing" of the shoulder is almost certain to follow.

Fig 1



Fig 2

Fig 1—Poor position postoperatively (five days). (a) head to unaffected side (b) hip to unaffected side (c) too many pillows (under shoulders) (d) buttock weight not evenly distributed.

Fig 2—Exercise 1

At Bellevue Hospital during the past eighteen months, a comprehensive rehabilitation program for the postoperative tuberculosis patient has been carried out as a cooperative program of the Chest Service and the Department of Physical Medicine and Rehabilitation to effect a physiologically sound means of com-

Fig 3



Fig 4

Fig 3—Exercise 2

Fig 4—Exercise 3

bating residual disabilities. Since all patients were seriously ill with tuberculosis, the intensity and frequency of the therapeutic efforts had to be carried out with extreme caution.

The corrective therapy program has been divided into two distinct phases: the pre- and postoperative. The objective of the former is to "educate" and

strengthen the syneigistic muscles not directly affected by the operative procedure, so that they may take over, in so far as possible, the functions of the primary muscles which are temporarily disabled. Further, the patient is carefully trained in the techniques of diaphragmatic breathing, which is essential in the postoperative stage. This is combined with emphasis on proper posture—lying, sitting, and standing. Finally, the patient is taught the basic exercises which he will require postoperatively preoperatively. This program is initiated twice daily ten days before the scheduled operation is carried out. Teaching is greatly simplified by the use of mirrors. Within twenty-four hours after the operation, the second phase is started and is carried on until maximum possible rehabilitation is attained, which may be a matter of weeks or many months.

Fig 5



Fig 6

Fig 5—Exercise 4

Fig 6—Exercise 5

Corrective exercises are customarily divided into (a) passive—where the entire movement is carried out by the therapist, (b) assistive (active assistive)—wherein the patient does as much as he is capable of, and the therapist completes the indicated range of motion, (c) active—wherein all the motion is carried out by the patient himself, (d) active resistive—wherein "load" is added to the exercises in order to increase the power of the muscles.

The routine of exercises used in postoperative surgery is as follows:

PREOPERATIVE STAGE

Position lying Bed mirror tilted over patient

EXERCISE 1—Emphasize correct bed posture (a) *Level pelvis*—avoiding any rotation or “hitting” of one hip (b) *Level shoulders*—which are held parallel to the level hips. The shoulders must be in normal relaxed position and not be elevated (c) The head, in cases of impending thoracoplasties, is held in position of lateral bending to the operative side. In thoracic surgery other than thoracoplasties, where the scaleni muscles are not severed the head is held in normal position, at a right angle to the trunk.



Fig 7—Exercise 6

EXERCISE 2—Pelvis and shoulders level, head normal. Patient places left hand on chest, right on abdomen. He is instructed to breathe abdominally (diaphragmatically), the right hand moving with each inspiration and expiration. Patient is taught not to move the chest or the left hand, thus striving to avoid as far as is possible thoracic breathing.

EXERCISE 3—Position—supine. Patient bends trunk laterally toward non-operative side. He is instructed to breathe diaphragmatically with the “operative” side only. Postoperatively this exercise is most important in the hastening of mobilization of the affected side of the diaphragm and thus help avoid paradoxical breathing in thoracoplasties and speeding pulmonary re-expansion in excisional operations. Incidentally, this exercise induces the cough reflex, which is of considerable importance in evacuating the pathologic contents of the bronchi.

EXERCISE 4—Active head extension, position—supine. Pelvis, shoulders and head held level. With chin depressed patient presses head and neck backward for count of two. Relax for count of three. Repeat sequence three times. Objective to strengthen the primary and synergistic extensors of the head and the cervical spine (upper fibers of trapezius, splenius, longus colli, etc.).

EXERCISE 5—Active shift of head to operative side basic supine position. Head is moved laterally (like a Bah dancer) only to one side. The head, held at right angles to the body, is slid to the operative side for a count of two, returned to normal position for a count of three. Repeat alterations three times. *Object* since (in thoracoplasties) the scalenus group will be permanently sectioned, the corresponding group (of the unoperated side) will tend to form

Fig 8



Fig 9

Fig 8—Exercise 7

Fig 9—Exercise 8

contractures, deviating and rotating the upper spine. By stretching these muscles and also by strengthening the synergists of the affected scaleni, these potential deformities are anticipated and may usually be greatly limited.

EXERCISE 6—Active adduction and depression of scapula (unilateral), patient supine. Therapist places his hand under the scapula. Patient is now instructed to press backward medially and downward on the supporting hand.

Object to teach the patient the specific coordinative exercise that will be required postoperatively to restore the function of the adductors and depressors of the affected scapula (rhomboids, mid and lower fibers of trapezius, latissimus dorsi, etc.) This exercise incidentally stretches the pectoralis muscles, combating tendency to forward rotation of the shoulder. Repeat three times holding for count of three, rest periods of three seconds between exercises.

EXERCISE 7—Active assistive depression of “affected” shoulder girdle. *Position* supine. Forearm is flexed on upper arm, fingers on shoulder. Arm held at side of trunk. Therapist “fixes” shoulder girdle and flexed elbow joint with his fingers. Patient depresses the flexed arm downward, stretching the shoulder capsule, ligaments, and muscles. Hold for count of two. Relax, to normal position (do not raise shoulder above normal level) for count of three. Repeat three times. *Object* to stretch and strengthen the muscles of the shoulder girdle (latissimus, lower fibers of trapezius, pectoralis, etc.) and thus try to prevent postoperative “high shoulder.” *This completes the preoperative procedures.*

POSTOPERATIVE ROUTINE

Start with the seven basic exercises given preoperatively. Follow the same sequence, timing, and alternation. Continue.

EXERCISE 8—Abduction of the arm at shoulder. The procedure is active-assistive and unilateral. *Position* supine, arm of operated side is flexed at elbow and held close to chest. Therapist supports arm at flexed elbow and at hand. Patient is encouraged to abduct the arm without strain, the therapist assisting the active effort. The exercise is repeated three times with intervals of rest of three seconds. *Objective* to assure maximum possible abduction of the shoulder joint by preventing “freezing” and muscular atrophy.

EXERCISE 9—Active assistive rotation of the shoulder joint. *Position* supine, arm in same starting position as for Exercise 8. Therapist swings arm to maximum abduction (as in Exercise 8), then the arm is rotated at first externally then internally. Again the patient is encouraged to carry out this exercise to the point of tolerance, the therapist supplying the degree of assistance indicated. Repeat three times with periods of rest. *Objective* same as with abduction, to assure maximum mobilization of the joint and hasten rehabilitation of the muscles of the shoulder.

EXERCISE 10—Active assistive “horizontal” abduction and adduction of arm (unilateral). *Position* supine, arm flexed at elbow and abducted to 90 degrees if possible, or not, as close as is possible. The therapist supports the flexed arm the same as in the previous exercises 8 and 9. *Object* (a) to stretch the postoperative adhesions, and (b) to exercise the adductors of the scapula and the extensors of the arm during the abduction phase of this procedure. Frequency and time intervals are the same as in the previous exercises. The flexed arm is carried medially, striving to touch the opposite shoulder with the fingers.

Fig 10



Fig 11



Fig 12



Fig 10 —Exercise 3
 Fig 11 —Exercise 4
 Fig 12 —Exercise 5

EXERCISE 11—Active assistive elevation (flexion) of both arms *Position* supine Patient clasps hands with arms extended and resting on trunk He is instructed to elevate the arms slowly overhead to the point of tolerance, which may be quite limited in the early postoperative stages by the pain *Objective* mobilization of the shoulder joint and exercise of the muscles of the shoulder girdle and arm Repeat three times with customary rest periods



Fig. 13—Exercise 11

Throughout these exercises, it is essential to be on guard for substitution movements, i.e., elevation of the scapula, forward rotation of the shoulder, etc. It is desirable to achieve to physiologically normal joint movements.

The postoperative routine is diligently carried out in the lying position for a period of one week. At this time, if the condition of the patient permits, he is seated in front of a full-length posture mirror and the rehabilitative routine is continued as follows:

EXERCISE 12—Active unilateral flexion of head *Position* sitting with pelvis, shoulders, and head level Patient bends the head to the 'affected' side,



Fig. 10



Fig. 15



Fig. 14

Fig. 14 — Thoracic
Fig. 15 — Thoracic
Fig. 16 — Thoracic
Fig. 17 — Thoracic



Fig. 19

Fig. 18

Fig. 17

Fig. 17 — discharge 16

Fig. 18 — discharge 16

Fig. 19 — discharge 17

aiming to touch the ear to the shoulder. Avoid rotation of head. Hold for count of two. Return to normal position. Rest for count of three. Repeat alternations three times. *Objective* to strengthen the residual flexors of the affected side and stretch those of the symmetrical (unoperated) side.

EXERCISE 13—Active circumduction of both shoulders. *Position* sitting with arms flexed at elbows and held at sides of body, finger tips at shoulders. Patient carries flexed arms forward upward to right angles with the trunk, then the arms are abducted horizontally winging out to the sides of the body. Return to starting position. Repeat alternations three times with essential rest periods. *Objective* to keep the shoulder joints freely mobile and to stretch the matted soft tissues. If patient finds this exercise too taxing, limit the procedure to the affected shoulder only.



Fig. 20—Exercise 13

EXERCISE 14—Abduction and adduction of flexed arms. *Starting position* hands clasped back of neck, flexed arms winged at sides, shoulder high. Patient adducts flexed arms touching elbows at front of body then carries arms horizontally back to original position. Repeat alternations three times with usual intervals of rest. *Objective* to stretch the matted soft tissues and exercise, primarily, the adductors of the

EXERCISE 15—Adduction and depression of arms. *Basic position* hands clasped over umbilicus, arms held in front of body. Patient is

instructed to approximate the scapulae by contracting the adductor muscles. Repeat three times, with rest intervals. *Objective* to strengthen the adductors of the scapula and stretch the pectorals.

EXERCISE 16—Flexion and extension of the trunk. *Starting position* sitting erect, arms hanging loosely at sides, knees together. Patient slowly lowers trunk until head touches knees and hands reach floor. *Objective* mobilization of the spine and further stretching of the matted tissues.



Fig. 21—Patients doing group exercises which follow the basic routine as described. All patients shown had seven-rib thoracoplasties.

EXERCISE 17—Same as Exercise 11 (see above), but carried out from the basic sitting position.

EXERCISE 18—Because of the relatively strenuous nature of this procedure, it should not be utilized until the patient has regained maximum strength and vitality. With the patient in sitting position, forearm at right angles to upper arm and supinated, the patient carries the flexed arm posteriorly while the therapist applies resistance against the elbow. Return to starting position, repeat three times with rest periods. *Objective* to develop the pectoralis minor to actual hypertrophy in an effort to fill in the postoperative cavitation or the upper part of the anterior aspect of the chest wall.

COMMENT

During the past eighteen months we have observed the effect of a carefully planned rehabilitation program in patients with the apparently inevitable deformities which in 257 patients undergo thoracoplasty. There is a prevalent opinion

Fig 22



Fig 23

Fig 22—Case W. H. Classical scoliosis resulting from thoracoplasty in patient who did not receive rehabilitative measures. Patient had (1) ten rib thoracoplasty (2) pneumonectomy and (3) pectoral myoplasty (for bronchopleural fistula).

Fig 23—Case W. H. Range of motion obtained in same patient after pectoral myoplasty when first rehabilitative measures were instituted.



Fig 24—Use of bed pulley for stretching after pectoral myoplasty which closed a broncho pleural fistula.



Fig 25—Typical high shoulder due to riding of angle of scapula on the eighth rib. Cervical scoliosis also results from the mechanical interference.

Fig 26



Fig 27

Figs 26 and 27—Case T M. Patient received no thoracic thoracoplasty in 1936.

that scoliotic deformity does not, as a rule develop until many months after the operation. In our experience, the evidence suggests that this deformity often begins immediately after thoracoplasty. The more extensive the latter, the more rapid is the onset of the scoliosis. The remedial results obtained in 107 thoracoplasties and 180 excisional operations (with or without incidental limited thoracoplasties) were encouraging to the degree of justification of the effort and its continuation. It is recognized that the most conscientious and intensive rehabilitative routine may not always prevent the scoliosis. How-



Fig 28



Fig 29

Figs 28 and 29—Case V. O. Patient who received pre- and postoperative exercises with a severe rib thoracoplasty. These pictures were made one year postoperatively.

ever by evaluation of twenty seven nontreated compared to the treated series, considerable improvement in thoracic deformity (particularly in thoracoplasties), there was not the same range of motion of the corresponding shoulder (in some cases to the point of complete ankylosis), there was also not infrequent shirring of the chest wall with resultant disturbance of cardiorespiratory function. This discomfort due to extensive arthritis and experience has shown the rehabilitation period of many weeks, months, and years. The most important factor is paramount—not only the technique

but the importance of taking the exercises with planned regularity. To aid in this education program, a booklet illustrating and describing the exercises has recently been prepared for patients.

SUMMARY

1 During the past eighteen months, at Bellevue Hospital a carefully planned routine for rehabilitative care of postthoracic surgery cases has been in operation.

2 The results obtained by means of diligent corrective exercises have prevented, to a varying degree, scoliosis, "freezing," of the shoulder, matting, and atrophy.

3 Comparative study of nonrehabilitated cases emphasizes the advantage of these preventive and remedial measures.

4 Postoperative rehabilitation is an integral part of the total care of the patients who undergo thoracic surgery.

Photographs were taken by Mr. Stanley Simmons, photographer for the Department of Physical Medicine and Rehabilitation, New York University College of Medicine.

A RACK FOR UNDERWATER DRAINAGE BOTTLES

JOHN D. STEELE, M.D.
MILWAUKEE, WIS.

A SIMPLE, inexpensive rack for underwater drainage bottles is presented. This rack has the following advantages: the bottles cannot be tipped over, the fluid levels in the bottles and glass tubing are visualized easily because of the light behind the bottles, and the rack can be attached to a standard hospital bed temporarily (by means of hooks which fit over the bed springs)

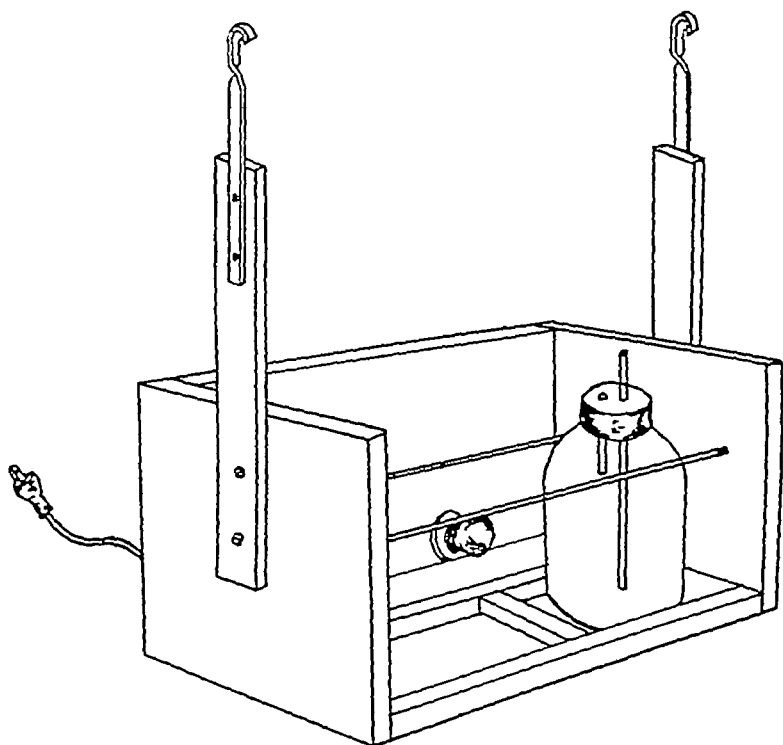


Fig 1

This rack can be constructed entirely of wood. However, the use of metal cross rods permits slightly better visualization of the fluid levels in the glass tubing. The $7\frac{1}{2}$ watt lamp also serves as a convenient night light. The back of the rack, above the lamp, provides a space for printed instructions to hospital attendants regarding care of the bottles.

In some types of hospital beds (usually inexpensive ones) there is sufficient clearance between the bed springs and the cross rods of the bed to attach the rack to wooden cross pieces which are clamped to the rods. This eliminates the necessity of unhooking the rack from the bed (when the springs are elevated) and allows the bed to be moved at any time without disturbing the bottles.

From the Department of Surgery, Marquette University School of Medicine.
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Announcement

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The 33rd annual meeting of the American Association for Thoracic Surgery will be held March 27, 28, and 30, 1953, in San Francisco, Calif. Headquarters will be at the Hotel Fairmont. Suitable accommodations also have been reserved at near by hotels, the Mark Hopkins, the Huntington and the Sir Francis Drake.

Requests for Hotel Reservations

These should be mailed to Dr. H. Brodie Stephens, Chairman of Hotel Committee, American Association for Thoracic Surgery, Room 200, 61 Grove St., San Francisco 3, Calif. The following information must be included: (1) two choices of hotels, (2) type of accommodation desired—single, double, twin, room or suite, (3) name of other room occupants, if any, whether wife or another doctor, (4) date and approximate hour of arrival, (5) date of departure, (6) return address.

Abstracts for Papers

Abstracts for the presentation of papers at the 1953 meeting must be received on or before Dec. 1, 1952, otherwise they will not be considered by the Program Committee. *Five, repeat, five* copies of each abstract should be sent to the Secretary of the Association, Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif. They should not be sent to the Editor.

The abstracts should contain from 200 to 250 words, and should accurately reflect the contents of the completed paper.

Applications for Membership

Completed membership applications must be sent to the Membership Committee, 1107 Clark Bldg., San Francisco 4, Calif., in order to be acted upon at the annual meeting.

All correspondence regarding membership applications should be addressed to Dr. Kent.

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Original Communications

TRAUMATIC SACCULAR ANEURYSM OF THE THORACIC AORTA

R. K. HOLLINGSWORTH, M.D.,* WILLIAM W. JOHNSTON, M.D.** (BY INVITATION),
AND JAMES F. MCCOY, M.D.* (BY INVITATION)
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TRAUMATIC saccular aneurysm of the thoracic aorta is apparently rare. Few cases are reported in the literature, no patients reported have survived more than eleven months, and all have been diagnosed at autopsy. This report is based on four patients, diagnosed during life, who have survived from a few months to seven years following their injury. In one case the development of the aneurysm was observed by serial roentgenograms. Correlating our observations with a study of the mechanism and pathology involved in traumatic rupture of the aorta has led to certain conclusions: (1) that this condition may occur far more frequently than is supposed, (2) that incomplete rupture of the aorta may possibly be diagnosed early and definitive treatment of the rupture instituted, (3) that if the aneurysm is already forming or has formed treatment to prevent further enlargement or later rupture may be carried out.

CASE 1—L. F., a 27 year-old white man, was admitted to the hospital on Sept. 29, 1949, with a history of having been involved in an automobile accident six days previously. He had been unconscious following the accident and hospitalized elsewhere. He had received multiple abrasions and lacerations, injury to the left forearm and hand, and severe confusion of the chest. There had been some coughing up of blood on the first day after injury. At the time of admission here the only complaint referable to the chest was occasional pain in the chest when coughing or on deep breathing.

Other than the residuals of the trauma, physical examination was essentially negative. Some dullness was noted on the left side of the chest posteriorly to about the level of the eighth rib. There was tenderness of the left wrist on motion.

Routine laboratory tests were within normal limits except the Kahn, which was reported positive 32 Kahn units. This test was repeated a week later with the same result. The spinal fluid Kahn was negative. A repeat Kahn in one month was reported positive, 8 Kahn units. Four days later the result was doubtfully positive, 0 Kahn units, and within three

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Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Read at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery, Dallas, Texas, May 8 to 10, 1952.

*Department of Surgery

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weeks the report was negative. In view of a negative history of a primary lesion and the rapidity with which the Kahn test became negative (no treatment was given), it was the opinion of our medical consultant that it was unlikely that this man had syphilis.

Roentgenogram of the left forearm and hand revealed a fracture of the ulna and of the proximal phalanx of the ring finger. A film of the chest on September 30 showed clouding over the left base, which was interpreted as fluid.

The fractures received appropriate treatment, and thoracentesis of the left chest produced about 75 cc of blood. In a few days the patient was ambulatory, but he remained in the hospital for treatment of his fractures. Repeat chest roentgenograms were made on October 4 and 11, and on the latter date there was no further evidence of fluid. (Discussion of all roentgenograms will follow the completion of the case history.) On December 13 a



Fig 1—Case 1. Saccular aneurysm as seen at operation.

repeat roentgenogram of the chest was made before the patient was to go on a Christmas pass. It was at this time that a definite mass was seen in the region of the aortic knob, and further studies, including fluoroscopy, were done. Pulsation of this mass was seen, but it could not be determined whether or not it was transmitted. Following his return to the hospital early in January, roentgenograms of the chest indicated that the mass had continued to enlarge, and angiograms were made. These showed conclusively that the mass represented an aneurysm of the first portion of the descending aorta.

Operation was advised, and on Feb 2, 1950, the chest was explored through the bed of the resected fifth rib. Except for adhesions of the lung to the aneurysm, the pleural cavity was free. These adhesions were carefully freed by blunt and sharp dissection and the aneurysm exposed (Fig 1). No pulsations could be seen and the wall felt quite firm. The

upper limit was approximately 5 cm. distal to the subclavian artery take-off, and it gave the appearance of a large bulge on the lateral side of the aorta. The aneurysm was well localized to the lateral wall, and it was felt that local covering with Cellophane would be sufficient. A flap of pleura was freed from the base of the entire aneurysmal area, and a piece of sclerosing type of Cellophane was sutured to the flap, completely covering the lesion. This in turn was covered by a sheet of non-sclerosing Cellophane to protect the lung. Closure was routine with one catheter as a drain, connected to an underwater seal. Recovery from the operation was uneventful, but discharge from the hospital was delayed so that the patient could receive physiotherapy for the arm and hand fractures.

Follow up has been regular at six month intervals and except for clearing of pleural reaction, there has been no change in the lesion. However, on Jan. 24, 1952, the patient was readmitted, complaining of a constant dull pain referred through from between the shoulders posteriorly to the precordium anteriorly. He had been doing heavy work running an air compressor for the past six months. Fluoroscopy and roentgenogram including angiogram, revealed no change in the lesion (Fig 3). Following three weeks' rest in the hospital he improved and was discharged, being strongly advised to seek less laborious work.



Fig 2.



Fig 3

Fig 2—Case 1 Admission roentgenogram, Sept. 30 1949 one week after accident. Note widening of mediastinum.

Fig 3—Case 1 Oct. 4 1949 Little change except clearing of pleural fluid.

Discussion—Fig 2 shows the initial chest roentgenogram. In addition to the evidence of pleural fluid, you will note the narrow band of increased density just to the left of the heart shadow. No significance was attached to this in the initial reading. Strassmann found that in eight cases with partial rupture of the aorta at this site the patients lived three or more hours, and at autopsy a tear was found extending through all coats of the vessel except the adventitia. There was dissection of blood along the aortic wall between the adventitia and media. In retrospect, we believe that this shadow represents widening of the aorta due to dissection. Strassmann's patients died of shock

or other effects of their injury. Our patient fortunately survived the immediate trauma, and in Figs 3 and 4 we see further developments. These are roentgenograms taken four days and eleven days, respectively, after Fig 2, and we see a change in this shadow, with the lesion becoming more localized at the site of the tear, and probably absorption of some of the dissecting blood. While this shadow was noted in the original roentgenographic report, its significance was still not recognized. However, in the film taken approximately one month later (Fig 5) there is little room for doubt. We felt that we were dealing with aneurysm, probably of the aorta, and that angiograms should be made. In spite of our advice and explanations of the danger involved, the patient insisted on waiting until after Christmas. On his return to the hospital early in January, roentgenographic studies indicated still further enlargement of the mass (Fig 6), and angiograms (Fig 7) proved conclusively that we were dealing with an aneurysm. Operation was advised and accepted. Fig 8 shows the roentgenographic appearance of the lesion two years after operation.



Fig 4

Fig 5

Fig 4—Case 1 Oct. 11 1949 The sac is beginning to form.

Fig 5—Case 1 Dec. 16 1949 Sac is enlarging

CASE 2—S L W, a 27 year old white man, was admitted to Kennedy General Hospital on Jan. 25, 1945. He had been injured in an ambulance accident in Paris, France, on Nov. 27, 1944. These injuries resulted in paraplegia from the level of L4. All laboratory reports were within normal limits. The history revealed that at the time of the accident a roentgenogram of the chest showed considerable wedging of the mediastinal shadow which could be due to mediastinal hemorrhage. Following a repeat film two weeks later it was reported that this mediastinal enlargement had disappeared. On Jan. 15, 1946, a routine chest roentgenogram was reported as showing a localized small density in the left superior mediastinum which was thought to be either a small dermoid cyst or lymph node enlargement.

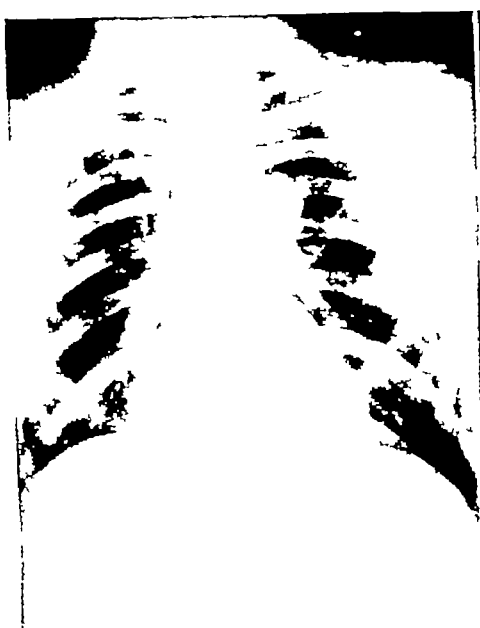


Fig 6



Fig 7

Fig 6—Case 1 Jan. 31 1950 Preoperative film Size of aneurysm has increased slightly

Fig 7—Case 1 Jan 13 1950 Angiogram showing aneurysm



Fig 8—Case 1 Jan 21 1952. Checkup film two years after operation.

(Fig 9) On May 25, 1946, an exploratory thoracotomy revealed this mass to be an aneurysm, 5 cm. in diameter, projecting from the lateral wall of the first portion of the descending aorta. The chest was closed, and recovery was uneventful. He was discharged from the hospital on Aug 7, 1946. There were no chest symptoms.

Discussion—From the record available we have no report of chest roentgenograms from December, 1944, to January, 1946. The mediastinal shadow appearing in the initial film cleared rather rapidly, indicating that it probably represented mediastinal hemorrhage. Just when the aneurysm first became visible or how fast it enlarged is unknown. It is suggested that in all cases of severe chest trauma, particularly if there has been any evidence of widening of the



Fig 9

Fig 10

Fig 9—Case 2 Preoperative film

Fig 10—Case 2 Induction film no evidence of aneurysm

mediastinal shadow, repeat roentgenograms should be made during the first two or three months. Fig 9 shows this lesion preoperatively. Fig 10 is the induction film taken in 1941. No aneurysm is visible. Fig 11 is a checkup film taken in August, 1951. No definitive treatment was given, and it would be interesting to know if it would have been wrapped with Cellophane had that material been available at that time.

This patient has remained well and without symptoms or evident enlargement for over seven years after his accident. But he is a paraplegic and has been relatively inactive. Occupation may play a part in the late development of symptoms or rupture.

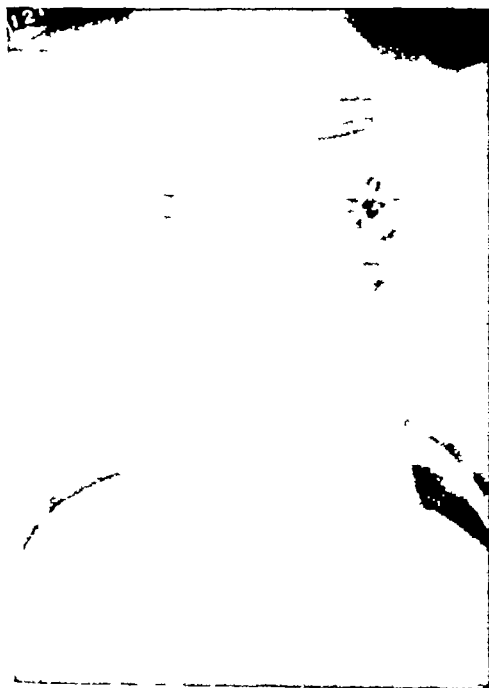


Fig 11—Case 2 Seven years after injury

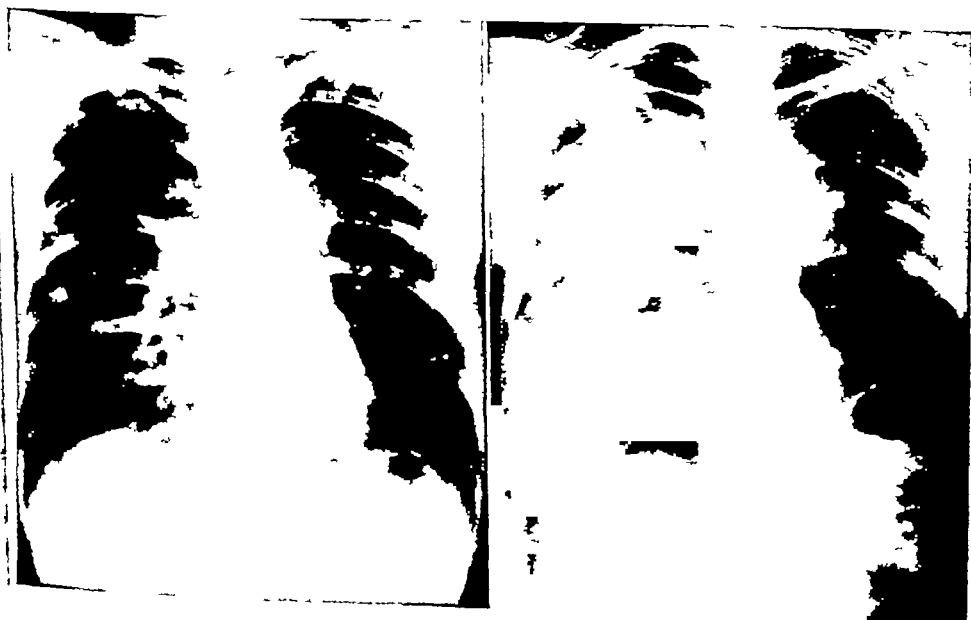


Fig 12

Fig 13

Fig 12.—Case 3 Jan. 30 1950 Routine separation film.
 Fig 13.—Case 3 June 10 1950 Last checkup five months postoperative. Note increase in size.

CASE 3—This patient was a 19 year old soldier whose lesion was discovered on routine separation chest roentgenogram in January, 1950 (Fig 12) On fluoroscopy there was some pulsation of the mass, but it could not be determined whether this was actual or transmitted. On barium swallow the esophagus was seen to be displaced slightly to the right and posteriorly. Physical examination was negative. All laboratory data, including serology, were negative.



Fig 14

Fig 14—Case 4 Jan 5 1952 Initial film posteroanteriorly



Fig 15

Fig 15—Case 4 Jan 5 1952 Lateral showing mass and fracture of sternum.

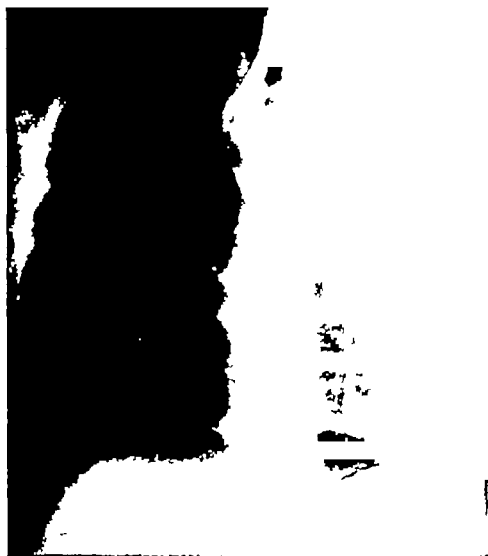


Fig 16—Case 4 Barium swallow

The only significant feature of his history was that he had been involved in a jeep accident approximately one year previously. No chest films were available from that accident. The chest roentgenogram on his admission to the army was thought to be negative, but it was not available.



Fig. 1—1 Case 4. Angiocardiogram eight and one-half seconds. B Ten seconds. C Thirteen seconds. Note dye pooled in aneurysm. D Seventeen and one-half seconds.

Left exploratory thoracotomy was performed on Feb 15, 1950, and a large sacular aneurysm of the first portion of the descending aorta was found. The chest was closed. On March 22, 1950, the left chest was again explored with the idea of excising the aneurysm. However, it was thought to be too extensive. Furthermore, the thinnest portion was so densely adherent to the pulmonary artery, bronchus, and vein, it was thought that dissection would be entirely too hazardous.

Postoperatively there was considerable difficulty in re-expanding the left lung, but he was finally discharged from the hospital on May 11, 1950. The last roentgenogram was in August, 1950 (Fig 13). It shows that this mass was definitely larger.

Discussion—Here is an otherwise healthy young man who had been in a severe accident one year previous to the finding of a large sacular aortic aneurysm. While no pre-accident roentgenogram is available, it is reasonable to assume that he had no such mass when inducted into the army, and we are accepting it as a true traumatic sacular aneurysm of the aorta.

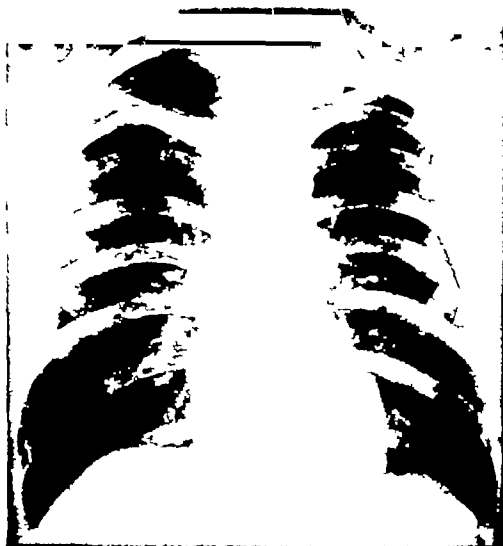


Fig 18—Case 4 Feb 21 1952. Note increase in size of aneurysm.

Cellophane wrapping was considered at the first operation and, had it been available, might have been used. The only follow-up available is contained in a letter and reported by Dr. Paul to the effect that one year after leaving the hospital he is apparently "all right." Further follow-up is important in this case, and every effort will be made to obtain it.

CASE 4—M. W. A., a 33-year-old white man, was admitted to Mt. Alto Hospital, Washington, D. C., on Dec 28, 1951. The chief complaint was substernal pain which was increased by swallowing. This had been present since an automobile accident on Dec 15, 1951, at which time he was struck in the chest by the steering column of his car. He was unconscious for about two hours, and suffered numerous minor cuts and abrasions. The substernal pain, aggravated by swallowing, had been constant. It was knife-like, and occasionally radiated through to the thoracic spine. Following one week of hospitalization he was discharged, returned to Washington, and had a chest roentgenogram taken at Arlington Hospital. He was told that he had a mass in the posterior superior mediastinum, and that this mass most likely represented a hematoma.

Physical examination was essentially negative except for tenderness over the sternum at the level of the third costal cartilage. The trachea was deviated slightly to the right. All laboratory examinations were within normal limits, except the urine and blood sugar. This man was a known diabetic.

Posteroanterior roentgenogram of the chest (Fig 14) taken on Jan 5, 1952, demonstrated widening of the superior mediastinum. The lateral film shows this mass to be somewhat posterior, and that there is a fracture through the middle of the sternum (Fig 15). A barium swallow revealed a mediastinal mass with displacement of the trachea and esophagus (Fig 16). Angiocardiograms revealed an aortic aneurysm arising from the left aspect of the transverse arch (Fig 17, A, B, C, D). A film taken on Feb 21, 1952 (Fig 18), showed a moderate but definite increase in the size of the aneurysm. A chest roentgenogram taken at Perry Point Hospital seven months previously was reviewed, and no evidence of a posterior superior mediastinal mass was noted.

Thoracic surgery consultation was obtained and a diagnosis of traumatic aneurysm of the aorta was made. No treatment was recommended and the patient was advised to have a chest roentgenogram once a month. The hospital course was uneventful and the patient was discharged on Jan. 31, 1952.

Discussion—There is no question but that this is a traumatic aneurysm. An interesting feature is the location, which is somewhat unusual. Here, instead of the usual indirect cause of aortic laceration (discussed later), we probably have a direct injury from a caving in of the sternum against the underlying aorta. The lesion showed definite evidence of increase in size during the hospital stay. Our knowledge of the ultimate outcome of this type of lesion without treatment will be enhanced by a careful long-term follow-up.

LITERATURE

Blunt force injuries to the chest may cause a variety of intrathoracic injuries to the heart or great vessels. Rupture of the heart, a heart valve or the aorta, purulent pericarditis, chronic adhesive pericarditis, mitral stenosis, and aneurysm of the heart are some of the results of trauma reported in the 126 cases collected by Bernstein.¹ He found that the first case of traumatic aneurysm was reported by Lesta in 1813. However, the report of this case would indicate that it was an aneurysm of the heart.

Traumatic rupture of the aorta is probably of more frequent occurrence than reports in the literature would indicate. Case reports by McDonald and Campbell,² Jaffe and Sternberg,³ Shennan,⁴ Landau,⁵ Kemp,⁶ Busse,⁷ and others have appeared from time to time. Strassmann⁸ reports that, from approximately 7,000 autopsies performed during the six years from 1936 to 1942 at the Chief Medical Examiner's Office of New York City, seventy-two cases of traumatic rupture of the aorta were collected. Of the sixty-one who had only one site of rupture all but three were in the thoracic aorta. Mennye⁹ reports a series of cases of aortic wounds with survival. Most were in the thoracic aorta but were caused by penetrating injuries. No late survival with aneurysm was mentioned.

Of much less frequency are reports of traumatic aneurysm of the thoracic aorta, first mentioned by Versalius¹⁰ over three centuries ago. Aschoff's case, cited by Stern,¹¹ was of the dissecting type, was in the ascending aorta, and was due to a gunshot wound. He survived the injury for four months. Leonard¹²

reports a patient 60 years of age who survived a crushing chest injury for sixteen days. At autopsy a dissecting aneurysm of the ascending aorta was found. Samson¹³ also reported a dissecting type of aneurysm of the descending aorta attributed to an accident five years previously. Hubener's¹⁴ case followed an automobile accident. It is impossible, from reading the case history, to be sure whether it was a saccular or dissecting type. Moxon¹⁵ (1870) reports one case and Albutt¹⁶ (1870) two cases diagnosed as traumatic aneurysm of the thoracic aorta. All three were diagnosed from clinical signs suggesting that they were located in the ascending aorta. Then traumatic origin was based on the fact that in each instance the patient was described as in excellent health before severe chest injury, and the aneurysm was diagnosed shortly thereafter.

Heller,¹⁷ in 1903, writes of a man 37 years of age who had a heavy mass fall on his chest. He died eleven months later, and autopsy revealed a saccular aneurysm of the first portion of the ascending aorta. Kaufmann¹⁸ reports seeing a "saccular aneurysm as large as a walnut in the thoracic part of the otherwise perfectly healthy aorta of a sturdy young man. It had developed some months after a severe fall." No further information is given. Shennan⁴ cites a case of a man of 57 years who died eight weeks after being knocked over by a bicycle. He died suddenly with a severe hematemesis. Autopsy revealed rupture of the thoracic aorta at the level of the fifth intercostal artery opening into an aneurysm which had in turn eroded into the esophagus and ruptured. Shennan calls this a false aneurysm, which classification is difficult to accept in view of the poor quality of the tissues which would of necessity form the aneurysmal wall in this area. Spearman and Nissen's¹⁹ cases were both due to gunshot wounds. Spearman's was in a 19-year-old boy who died from rupture and Nissen's in a 36-year-old man who was still alive after nine years. Stryker²⁰ in 1948 reports the case of an 18-year-old girl who was injured in an automobile accident five months before her death. At autopsy one large (4 cm by 5 cm) saccular aneurysm was found in the first portion of the aorta just above a cusp of the aortic valve. There was also a smaller defect on the opposite wall. Stryker also reports two cases from the records of the Department of Pathology of the University of Michigan in which death occurred shortly after severe trauma and in which there was incomplete tearing of the aorta. He believes this to be a first stage in the development of traumatic aneurysm.

DISCUSSION

Traumatic saccular aneurysm of the aorta may be due to blunt trauma to the chest or to penetrating wounds. This discussion is limited to the former. In addition, greater stress is laid on those lesions involving the first portion of the descending aorta. This is the most frequent site and the only one with which we have had any experience. Of no less importance are those occurring in the first portion or ascending aorta, and any further study of these lesions should, if possible, include both sites.

By definition²¹ a true aneurysm is one in which the sac is formed by the arterial walls, one of which, at least, is unbroken, and a sacculate aneurysm is a sacklike arterial dilatation which opens into the artery by an opening that is

small compared to the size of the sac. We believe that the aortic lesions under discussion are true sacculated aneurysms. They are a result of injury to that organ. The mechanism of formation is in direct relation to the degree of disruption of the aortic wall. Complete rupture means instant death. If the intima only is torn, a dissecting aneurysm may form or healing may occur, as Peery² has shown in cases of spontaneous rupture. If both the intima and media are ruptured, leaving only the adventitia, and death does not occur from other results of the accident, an aneurysm may form. To understand the mechanism of the formation of these aneurysms, therefore, one must study aortic rupture.

Most authorities believe that the arterial system, if healthy, is able to withstand, without rupturing, any conceivable increase in blood pressure which can be produced by a strongly contracting left ventricle during great temporary physical or mental strain. Klotz and Simpson²³ were unable to rupture the aorta of individuals from 20 to 40 years of age with pressures up to 1,000 mm. of mercury, while Oppenheim²⁴ reported rupture at a pressure of 3,000 mm. of mercury. It is therefore difficult to accept the theory that an increase in intra-arterial pressure causes a bursting rupture from within. However, it must be borne in mind that a *sudden* change in pressure will have greater effect than a continuous force,²⁵ and it is possible that this is a contributing factor. Obviously, other more important factors must be considered, and several theories have been advanced.

It is important, at this point, to consider the usual site of rupture. All reported aneurysms and practically all ruptures were at one of two sites, the first portion of the ascending aorta or the beginning of the descending aorta just distal to the ligamentum arteriosum. Strassmann's series, the largest reported, showed that in thirty-eight of sixty-one cases the rupture was in the latter location. As both of these sites are also those of predilection for spontaneous dissecting aneurysm, it must be agreed that they represent "weak" areas in what is usually considered a "tough organ." Both the first portion of the ascending aorta and the beginning of the descending aorta are comparatively fixed points, the former by the fibrous union between the aorta and the pulmonary artery, and the latter by the ligamentum arteriosum and the large vessels.

According to Warfield²⁶ the mechanism in injuries to the heart and the proximal portion of the aorta or the root is that in full inspiration sudden compression of the chest finds the heart caught and held between the inflated lungs. A violent blow on the chest, whether over the heart or not, may injure the heart, particularly if it is in diastole. It may rupture the myocardium, a valve, or the root of the aorta. Peery²² states that the valve commissures exert a rocking force and pull on the aortic wall as they are driven shut in diastole by the column of aortic blood, and this local force is probably the cause of the more frequent occurrence of tears at this point. The fact that the tears are usually transverse to the axis of the valve commissures adds weight to this hypothesis. While he was speaking particularly of spontaneous tears, Peery's theory appears valid in conjunction with that of Warfield in cases of traumatic rupture in this area.

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Strassmann believes that the majority of traumatic ruptures develop at the isthmus of the aorta because of the fact that the aorta is narrowed and relatively fixed at this site by the attachment of Botallo's ligament. This might render this portion of the vessel more subject to overextension or twisting or sudden increase in intra-arterial pressure.

According to Hass,²⁷ "whenever one part of the body is decelerated at a rate which is different from that of another part the connection between these parts is placed under stress which is proportional to the rates of deceleration." Rice and Wittstruck²⁸ have described their concept of the mechanism and shown it diagrammatically as reproduced in Fig 19. According to these authors, "in linear deceleration of the thorax, the central portion of the descending aorta, being the least rigidly bound, is snapped forward by the momentum of the deceleration force and the mass of the aorta's content of blood. This force is irregularly absorbed by the elasticity of the vessel and the thoracic contents the upper portion of the aorta is most rigidly fixed by the great vessels of the arch and the ligamentum arteriosum. This portion decelerates at the same rate as the whole body, while because of its less rigid fixation the descending aorta decelerates at a different rate. This difference constitutes the force which is concentrated at the point of maximum fixation."

It has been suggested that the area just distal to Botallo's ligament, being the usual site of coarctation, may be an embryologically weak point in the aorta, but no substantiating pathologic evidence has been shown. According to Abbott,²⁹ weakness at this point is usually associated with other vascular abnormalities.

Stryker²⁰ gives the only complete pathologic description of a traumatic aneurysm. Fig 20 is a cross section of the larger aneurysm in his first case. "The interrupted elastic fibers formed a tangled mass at the edge of the lesion, and there were scattered individual elastic fibers and small masses of fibers in the walls and floor." There was an organizing thrombus attached to a portion of the wall. An additional interesting and important pathological finding was the presence of subacute bacterial endocarditis.

TREATMENT

Obviously, from this small series of cases, no dogmatic statement regarding the proper treatment of these serious lesions can be made. As a matter of fact, with each case encountered, a different problem was presented, and we became more confused. Out of this confusion we have attempted to draw some valid conclusions but at this point we can only theorize and then place the matter before you for discussion and further consideration.

The treatment followed will vary with the stage of development of the aneurysm. Arbitrarily we have divided this into three stages. We believe, with Stryker, that there is a first or pre-aneurysm stage. Here we have the laceration of the inner two coats of the aortic wall and a varying degree of dissection. That stage is well demonstrated in Fig 2. Strassmann showed this in the eight cases of his series who had incomplete rupture. If, at this stage,

the patient's condition permits, exploratory thoracotomy with suture of the lacerated aorta may be feasible. Dshanelidze,³⁰ Blalock,³¹ Elkin,³² and Beattie³³ have all reported successful suture of a lacerated aorta. The difficulty here is twofold: (1) early diagnosis is essential and admittedly difficult, (2) unfortunately many, if not most, of these patients will be in serious condition from other injuries and not immediately operable. The duration of the first stage is undoubtedly short.

Referring to Fig. 3, taken two weeks after the accident, we see little change from Fig. 2. However, in Fig. 4 there is definite beginning formation of a sac. We assume from this that the first stage in this case continued for approximately two weeks. Should suture of the laceration not be possible, wrapping the area with Cellophane of the Polythene type, as recommended by Poppe and de Olivera³⁴ for syphilitic aneurysms, is suggested. This might prevent formation of the sac or at least produce enough reaction to limit its size.

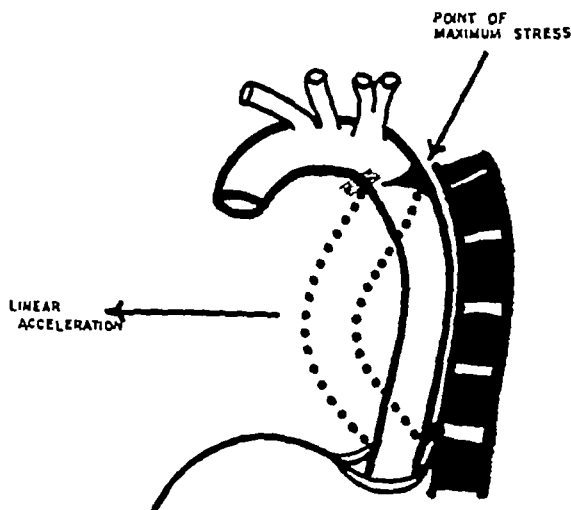


Fig. 19.—Diagrammatic representation of the mechanism of laceration of the healthy aorta by indirect violence (From Rice and Wittstruck, *J. A. M. A.*, Nov. 1951).

During the second, or developmental stage, as exemplified by Figs. 4 and 5, operation should be performed as soon as the condition of the patient permits. Here one of two procedures might be carried out. From the evidence available the wall of the sac consists of the adventitia, the opening into it varying with the size of the laceration. While the sac is still small it might be possible to resect it and then suture the laceration. A more conservative approach would be cellophane wrapping. This should prevent further increase in size and reduce the possibility of rupture.

The third, or late stage, will comprise those cases seen months or years after the accident, most probably seen on roentgenograms taken routinely or for other causes, as in Cases 2 and 3 of this report. There may possibly be symptoms present as in Nissen's case reported eleven years after a bullet wound received in World War I. While the actual cause of this aneurysm was different

from those under discussion, it gives us an insight into possible late results and thus might well influence the choice of treatment. Because it represents the longest known survival of a patient with a traumatic aneurysm, we feel that a brief mention of the symptoms is in order. They consisted of pressure feeling in the chest which increased to dyspnea on exertion, some cough with scanty expectoration, pains which radiate to the left arm, and disagreeable sensations in the area of the heart. These symptoms had increased considerably during the preceding two years, and Nissen felt that this was probably due to an increase in size of the lesion.



Fig. 20—Section through saccular aneurysm elastic tissue stain. See text. (From Stryker *Am. J. Clin. Path.* Feb. 1948.)

Whether or not surgical therapy should be recommended in this stage might depend on three factors: (1) the age of the aneurysm, (2) the age and occupation of the patient, and (3) the presence or absence of symptoms. Certainly with an aneurysm of considerable age, symptomless and in a patient with a more or less sedentary occupation, one might advise against surgery. But just when can a traumatic aneurysm be deemed old enough to be considered stable? In Case 3 we see rather marked enlargement, following exploration of the chest, in a lesion over a year old. Here occupation enters into the picture. Heavy labor is much more likely to be a cause of enlargement, symptoms, or even rupture, than light desk work. We believe very heavy labor to be contradicted, regardless of whether the lesion is treated or not. For the various gradations of work between that and a sedentary life, active treatment must be given very serious consideration. It is not enough to advise frequent chest roentgenograms, watching for a change in size, because any change may be so sudden and extensive as to quickly carry the case beyond help. Case 2 has gone seven years

without symptoms or change in the size of the aneurysm, but the patient is a paraplegic and has perforce led a sedentary life. Symptoms, at present, call for surgical interference. Nissen,¹⁹ in 1927, suggested that surgical interference should be considered in his case and should consist of transplantation of fascia around the wall, or, if the stem of the sac was small, extirpation.

The conservative treatment consists of Cellophane wrapping. We have no knowledge of the long-term results with this substance in this type of lesion, but the results in some syphilitic aneurysms are encouraging enough to warrant a thorough trial. Our patient has now gone two years with no increase in size of his lesion (Fig 8), but he has recently been hospitalized for study because of symptoms which conceivably could be due to the aneurysm. He had been operating a large pneumatic drill for the past six months, which may have caused his symptoms even if he had no aneurysm. Extensive studies, including an angiogram, were negative. He will report to us later after doing lighter work.

In patients in whom the lesion is diagnosed at an exploratory operation, we feel that wrapping should be done. It adds little to the procedure, and may prevent future trouble.

With our present meager knowledge of all phases of these lesions, we do not feel justified in recommending the more radical procedure of resection and grafting. Few clinics in the country have the blood vessel bank needed, the procedure is a formidable one, and until the more conservative treatment has been definitely proved inadequate, we believe that resection will be rarely indicated.

CONCLUSIONS

With the many crushing blows encountered in the ever-increasing accident rate, and with improvement in diagnosis and treatment of intrathoracic lesions, the early recognition of traumatic aneurysm becomes more and more important.

We would like to point out that a small density following parallel and outside the descending aorta was the first sign noted in Case 1. In the beginning, due to hemothorax, we interpreted the parallel density as pleural blood or fluid and no more significance was attached to it. As the shadow became larger and more dense, the exact meaning was not known until the outlines began to become sharp, and then aneurysm was considered.

In the development of densities along the left paramediastinum between the transverse aorta and hilum, many different causes must be considered. Katz and Reed²⁰ point out the possibility of paramediastinal pleural effusion which we first suspected. However, lymphoblastoma and superior and posterior mediastinal tumors, in various stages of growth, may give a similar picture, as seen in the first and third cases.³⁰ We have recently seen a case of a benign thymoma which presented a shadow almost identical to these two cases (Cases 1 and 3).

It is interesting to note that three of the four cases presented here have almost identical roentgenographic findings. This may be of aid in future diagnosis of this type of case.

Angiograms have been of great value in making a correct diagnosis³ when the condition of the patient permits. Fluoroscopic examination may or may not give additional information, but we feel it should be used as an adjunct diagnostic aid.

We believe that active treatment is indicated in most cases if it can be accomplished without unduly adding to the risk, and, until more is known of the ultimate outcome under normal conditions of living, this should consist of Cellophane wrapping. As mentioned earlier, our knowledge is too meager to be dogmatic, but it is logical to assume that no surgeon could sit idly by, watching an aortic aneurysm grow larger and larger, unless his considered judgment dictated that the risk of surgery was far greater than the lesion per se.

That a great deal of further observation and study is required is obvious, and it is to be hoped that the reporting of this small series of cases will stimulate this study. Such a study should include (1) extensive autopsy study of cases following severe chest injury, with particular reference to aortic trauma when death has been delayed and was not due to the aortic lesion, (2) autopsy study in all possible cases who die suddenly months or years following severe chest injury, and (3) careful roentgenographic follow-up of all patients who have severe chest trauma but in whom no immediate residuals are evident. It is probable that many serious internal injuries are unrecognized, the obvious injuries such as fractures, etc., directing attention away from the "silent" injury. Thousands of people are injured daily. There should be plenty of material for study.

We are indebted to Dr Felix Hughes and the Kennedy General Hospital, Col John Paul and the Walter Reed Hospital, and Dr Louis Alpert and Mt Alto Hospital for the use of Cases 2, 3 and 4, respectively, to Dr Walter Stryker, Dr S E Gould, Editor, *American Journal of Clinical Pathology*, and the Williams and Wilkins Company for permission to reproduce Fig 20, to Dr Walter G Rice, Dr Kenneth P Wittstruck, and the *Journal of the American Medical Association* for permission to reproduce Fig 19, to the Department of Medical Photography at Mt Alto Hospital for the figures of Case 4, and to the Veterans Administration Regional Office at Albany, N Y, for the follow up on Case 2.

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DISCUSSION

DR DENTON A. COOLEY, Houston, Texas—Despite the fact that numerous procedures have been suggested and advocated for treatment of aneurysms of the aorta, for the most part the results of such surgical techniques have proved to be unsatisfactory. With regard to the cases just presented, it appears worth while to call attention to a more direct and definitive technique in management of such lesions, emphasizing a fundamental principle of surgery, namely, the excision of destroyed tissue.

In experience with a small group of patients with aneurysms of the thoracic aorta and its tributaries has emphasized to us the practicability of excisional therapy in aneurysms so situated.

(Slide) A patient aged 37 years with coarctation of the aorta was admitted to the Johns Hopkins Hospital early in 1950 because of severe pain and pulsations in the right supraclavicular region. The coarctation was successfully resected by Dr Blalock. About eight weeks later the patient was readmitted for exploratory thoracotomy on the right side.

At operation an extensive saccular aneurysm of the first portion of the right subclavian artery was excised. Encouraged by the result obtained from excision of a large aneurysm, I used this technique on a patient with a syphilitic aneurysm of the innominate artery.

(Slide) The aneurysm arose from the arch of the aorta. The broad base of the aneurysm was occluded with a Crafoord clamp, and the aneurysm was excised. Lateral aortorrhaphy was done with a double row of silk sutures. The aortic tissue in this patient with syphilitic aneurysm was tough and leathery and held sutures well.

(Slide) In this patient, a 54-year-old man, there was a syphilitic aneurysm of the ascending aorta and of enormous size. The lesion was completely excised, and lateral aortorrhaphy accomplished to the 10 centimeter aortic defect. This patient died eighteen hours later, probably of cerebral anoxia, most likely an anesthetic complication related to the bilateral open thorax at operation. The first and second patients are alive and doing well.

In reported analyses of large series of aneurysms of the aorta, one is struck by the alarmingly high expected mortality. This feature of the problem places aneurysm, together with carcinoma, in the group of highly fatal diseases. An aggressive approach to such lesions by thoracotomy and attempted removal therefore is justifiable. An important feature in our cases of aneurysmectomy was a relatively pliable and elastic aortic wall suitable for suture. This is characteristic of syphilitic aneurysms, particularly in the relatively young patient. A traumatic aneurysm, such as illustrated by Dr. McCooey, and similar to my case of subclavian aneurysm, may likewise occur to an otherwise healthy aorta, and would, therefore, be suitable for suture techniques. On the other hand, aneurysms of an arteriosclerotic nature are probably not amenable to this type of therapy.

DR RALPH A. DETERLING, JR., New York—We have been fortunate at Presbyterian Hospital in New York to have the pioneering efforts of Dr. Blakemore lead us through various evolutions of treatment of aneurysms of the aorta. As has been mentioned, most of those of the thoracic aorta that we encounter are syphilitic. With this type the wall of the aorta adjacent to the lesion may lend itself very nicely to suture technique. Dr. Blakemore and those of us who have applied plasticized polythene or fibrosing Cellophane to the actual sacs have considered our overall results with this technique not too satisfactory. We know of several cases in which the aneurysm has been fairly well controlled, but the aorta has ruptured above the area where the Cellophane was applied. This is a hazard which certainly should be avoided if possible. Consequently, the usual treatment of choice at Presbyterian Hospital has been the installation of fine wire into the sac. I am speaking now of the saccular type, such as were presented today. Our experience with this method has been extremely good, the mortality being essentially zero. The pulsatile character of the sac, pain related to the bone erosion, and the actual size of the aneurysm has definitely decreased following application of between two and five hundred feet of wire. In some cases several applications of wire may be needed.

Dr. Blakemore himself prefers an apparatus which converts alternating current to direct current to permit electrothermal coagulation in the sac. He instills a specially insulated, flexible fine wire into the sac, which he heats at 80° C. for about ten seconds. This causes a coagulum to form on the wire mesh and perhaps produces a much quicker inactivation of the sac. I have not used this particular apparatus, but have relied mainly on 32 gauge steel wire. My results in a small series of cases have been very satisfactory also. Recently, I have been looking for cases such as were reported here in which we might be able to resect the sac and use a preserved homograft. I feel that any other method than actual restoration of a normal linear channel of blood flow is somewhat short of what we would like to achieve.

DR OSLER ABBOTT, Atlanta, Ga.—I should like to speak on three points. Our feeling with regard to aneurysm has been that whenever possible they should be resected. When they cannot be resected then we use a combination of Cellophane wrapping and wire, except in the ascending aorta, and the arch of the aorta, because of the frequency with which wiring has produced fatal cerebral involvement phenomena. The second point I would like to bring out is the symptomatology due to stimulation of the adventitia of the aorta in the traumatic

cases. These patients will frequently have burning pain, particularly during the night, quite similar to that of myocardial infarction. The third point I would like to mention is to try to clarify rather than confuse the situation with regard to Cellophane. The DuPont Company notified us quite a while ago, and I know that Dr Poppe had considerable correspondence with them, to the effect that they wish to be of all help possible to the medical profession, but are not a pharmaceutical house, they have now discontinued making, or stopped supplying as such, reactive and nonreactive Cellophane. What we have done is to follow the work of Dr Hufnagel and make our Cellophane nonreactive by soaking it long enough in alcohol. Then we make a supersaturated solution of diacetyl phosphate powder in toluol. We then paint this material under aseptic precautions on the Cellophane that we want to make reactive, then put it in zephiran for twenty minutes. There is no question that the reactivity is far superior to any that we have used before.

DR. ROLLIN A. DANIEL, Nashville, Tenn.—We have quite recently had the opportunity to operate on a Negro woman 67 years old, with a large aneurysm of the ascending aorta, with erosion of the sternum and marked tracheal obstruction. She had been unable to lie in bed for several weeks before the time she came to the hospital.

(Slide) We excised the aneurysm at its base. The patient expired four hours after she was returned to bed, of cardiac arrest. Resuscitation was possible, but another arrest occurred about an hour later, and another, and she expired. There are a few points, however, which I think may be worth mentioning. This slide shows an area where the aneurysm was dissected and the roughened area. This was an arteriosclerotic aneurysm, there was much calcium in the wall of the aorta. We dissected gradually and found it had a pedicle about two inches in diameter, considerably smaller in diameter than was at first realized.

(Slide) This depicts the suture line after excision of the aneurysm. The fact that she died leaves unanswered any question as to the fate of this suture line. Two other points which I think may be helpful, however, are first, the exposure, which was very good, obtained by transecting the sternum and crossing the anterior intercostal spaces, and then dividing the cartilages on each side. After they were divided the sternum protruded far forward. Second, just as the clamp was placed across the pedicle, the aneurysm was opened at its apex and clots and bright blood in small amounts from the aorta were evacuated, at the same time the clamp was tightened. I think that cutting through or fracture of the base of the aneurysm would have been more likely had the sac not been opened widely before the base of the aneurysm was clamped.

DR. MCCOOEY (Closing)—I wish to thank Dr Cooley, Dr Deterling, Dr Abbott, and Dr Daniel for their excellent contributions to this presentation. Our conclusion is that it is obvious further observation is necessary, and it is hoped that the recording of this small series will stimulate such a study.

HEALING OF EXTENSIVE CARDIAC WOUNDS

COLIN G THOMAS, JR, M D (BY INVITATION), CHAPEL HILL, N C, AND
SIDNEY E ZIFFREN, M D (BY INVITATION), IOWA CITY, IOWA

THIS study was undertaken because it was evident that continued progress in surgery of the heart would depend upon greater knowledge of the consequences of cardiomy. Little information was available regarding the formation of mural thrombi and subsequent embolization following incisional wounds, neither was information available regarding dehiscence of the wound, aneurysm, or rupture of the site. Accordingly, the investigation was designed to study the healing of the myocardium following extensive incisional and excisional wounds of the anterior wall of the left ventricle.

From clinical practice it is known that cardiomy is feasible, and considerable experience has accumulated in surgery directed toward valvular diseases of the heart, removal of foreign bodies, and repair of stab wounds. In these instances the defects of the ventricular wall have been relatively small, and healing has usually been without complication. However,¹⁴ an aneurysmal dilatation has been noted ten months following cardiomy for aortic valvular disease, and in another instance¹⁰ an embolus to the brain followed the repair of a stab wound. No long-term analyses have been reported regarding the course and end results of myocardial repair.

Cardiac muscle is a peculiar type of striated muscle and exhibits only slight capacity for regeneration.¹¹ Consequently, union is a function of the development of a fibrous scar strong enough to bridge the defect without yielding to intermittent intracardiac pressure.

From the general principles of wound healing, the course of myocardial repair may be fairly well anticipated.⁷ An initial "lag" period of several days would be followed by a second phase of increasing strength due to the rapid proliferation and slower maturation of the fibroblast. In structures with a high tensile strength, the third phase of increasing strength coincident with the maturation and increase in amount of collagen extends over weeks rather than days. Ultimately, the tensile strength of a wound is almost entirely a function of the number of fibroblasts, their maturation, and the adequacy of collagen formation. Scar tissue, however, is seldom as resistant to tension as is normal tissue.^{5, 13} Thus, if tension is present in any degree, the cicatrix may prove to be a vulnerable point in that area subjected to such pressure or tension. The heart muscle is unique in that it is constantly undergoing contraction and relaxation, and yet is at all times under tension.

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Read at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery, at Dallas, Texas, May 8-10, 1952.

On a theoretical basis, with the knowledge of wound healing as described and the changes that may result in a scar from prolonged tension, some attenuation or cicatrix joining cardiac muscle can be anticipated. Furthermore the physical forces are such that under certain circumstances the resulting scar would not only become attenuated, but possibly rupture or develop an aneurysmal defect. A somewhat analogous situation exists following coronary occlusion and subsequent myocardial infarction in which both rupture and aneurysm are known to occur. The development of such an aneurysm closely follows the occurrence of myocardial infarction, appearing within a week to several months after the acute episode.¹

Relatively few experimental studies have been directed toward this problem. Those by Elsberg³ in 1899 concerned the healing of heart wounds in rabbits, and disclosed the development of a scar that did not interfere with cardiac function. His observations extended over a four-week period, and in only one instance was the scar noted to be "thin and bulging." More recently, Carter observed broadening and thinning of the cardiac wound following excision of portions of ventricles.² These observations extended over a period of six months.

METHODS

Adult mongrel dogs weighing between 10 and 25 kilograms were utilized as the experimental animal. Employing intravenous pentobarbital sodium for anesthesia in conjunction with an intratracheal airway, thoracotomy was performed through the left fifth interspace. Ventilation was maintained manually while the chest was open. After dividing the pericardium parallel and medial to the left phrenic nerve exposure of the anterior surfaces of the right and left ventricles was obtained. The heart was bathed periodically with 2 per cent procaine to reduce irritability and to maintain moist surfaces. One suture was placed through the apex of the heart to facilitate control. Two groups of experiments were then carried out.

1. An area measuring approximately three by four centimeters, including the entire thickness of the left ventricular wall, was excised. This area was delineated by ligating an inferior twig of the descending branch of the left coronary artery and accompanying vein. The region or subsequent ischemia measured approximately two by three centimeters on the anterior surface of the left ventricle. This area plus the surrounding three to five millimeters of cardiac muscle was then excised attempting to divide the myocardium at right angles to its epicardial surface. The ligation of these small vessels usually does not produce an infarct,^{1,2} but serves merely to outline that area in which circulation might be temporarily impaired by interruption of these vessels at the time of excision. Incisions in this area are inferior to the papillary muscles and do not encroach upon the interventricular conduction mechanism. The resulting defect in the ventricular wall was closed by interrupted through and through sutures of No. 000 black silk. These sutures were placed approximately three-fourths to one centimeter apart and from one to one and one-half centimeters from the wound margin, passing through the epicardium, myocardium, and endocardium. The average length of the closed incision on the epicardial surface was between three and four centimeters, requiring five to six sutures for closure. Fifteen dogs were available for periodic examination during the course of wound healing, with examinations made over a period of forty-six months.

2. A linear incision was made in the left ventricular wall parallel to the inter-ventricular septum beyond the gross arborizations of the ramus of the anterior descending branch of the left coronary artery. The incision was approximately four centimeters in

length. Twelve dogs were observed over a period of eleven months. In two additional animals a two centimeter incision in the same location was carried out. Closure was effected as described in Group 1.

Since the techniques employed for limiting blood loss are not pertinent to this discussion, they will be described only briefly. Three methods were utilized. (1) Excision of the ventricular wall was performed with the blood loss being proportionate to the time required for wound closure. (2) Underlying mattress sutures were placed prior to incision of the myocardium and subsequently removed after closure of the cardiac defect. (3) In the majority of animals the superior and inferior venae cavae were clamped, occluding the return flow of blood to the heart, except that through the azygos vein and coronary sinus. This last method was found to be the most expedient as far as diminishing blood loss was concerned, and also decreased manipulation of and trauma to the heart. Wound healing followed the same general pattern regardless of the technique employed.

In the pilot experiments, serial electrocardiograms were procured during all phases of the procedure as well as early and late in the postoperative period. The animals were sacrificed at periodic intervals under pentobarbital sodium anesthesia. In each instance the heart was observed directly for evidence of cardiac aneurysm.

CHANGE IN DEPTH OF SCAR FOLLOWING EXCISION OF VENTRICULAR WALL

Depth of scar expressed as % thickness of
normal ventricular wall

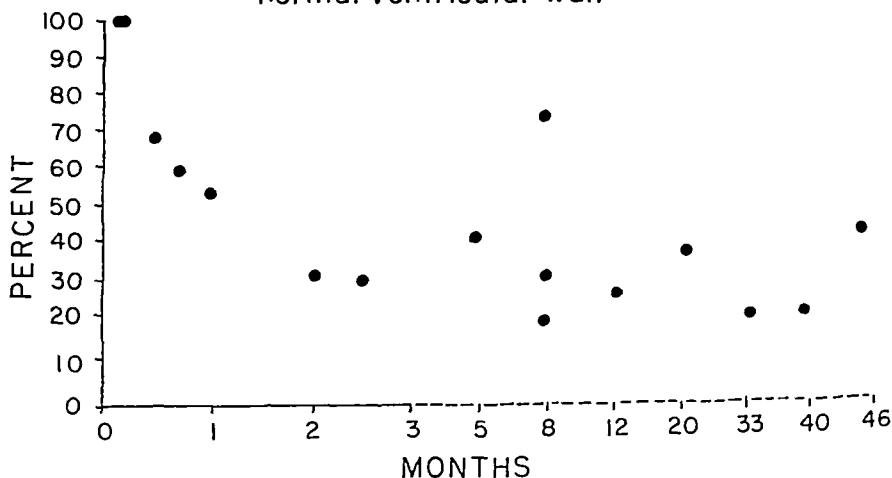


Fig 1

RESULTS

Group 1—With excision of a portion of the ventricular wall healing was characterized by the early production of a narrow scar measuring the entire thickness of the apposed surfaces. This depth of scar was maintained for approximately one to two weeks, and then rapidly decreased in thickness during the ensuing two weeks so that at the end of one month the cicatrix joining cardiac muscle measured only 50 per cent of the thickness of the adjacent normal ventricular wall*. With increasing intervals of time there usually developed

* All measurements were taken from the midportion of the scar which was usually thinnest, with graduation toward extremities.

attenuation until the scar measured 30 per cent of the ventricular wall (Fig 1) In some instances this developed as early as two months, whereas in others a relatively thick scar was maintained until the time of sacrifice at eight months The greatest attenuation was observed in those animals sacrificed at thirty-three and forty months (Fig 2)

Concomitant with early healing, there also occurred increase in breadth of the scar so that in its midportion it usually measured between nine and eleven millimeters These changes were observed as early as one week, and seemed to bear no direct relationship to the depth of the scar or the elapsed time (Fig 3)



Fig. 2.—Endocardial, epicardial, and cross sectional views of heart thirty-three months following excision of 2 by 3 cm. area of left ventricular wall.

Group 2—With linear incision of the ventricular wall the course of healing was essentially as observed in Group 1 Differences in degree were noted, however, viz., attenuation of the cicatrix was present but was less pronounced, and in only one instance was it less than 50 per cent of the thickness of the adjacent ventricular wall (Fig 4) There was also more variation in the maximum breadth of the scar, which varied from two to eleven millimeters Again there was no correlation with elapsed time or depth of scar In the two dogs in which two centimeter incisions were made in the same location, very little tendency toward attenuation was observed over a period of four and six months (Fig 4)

In all dogs the following features were observed to occur without regard to the particular procedure carried out

1 *Histologic changes*—Microscopic examination disclosed an initial death of that cardiac muscle made ischemic by wound closure This zone of necrosis

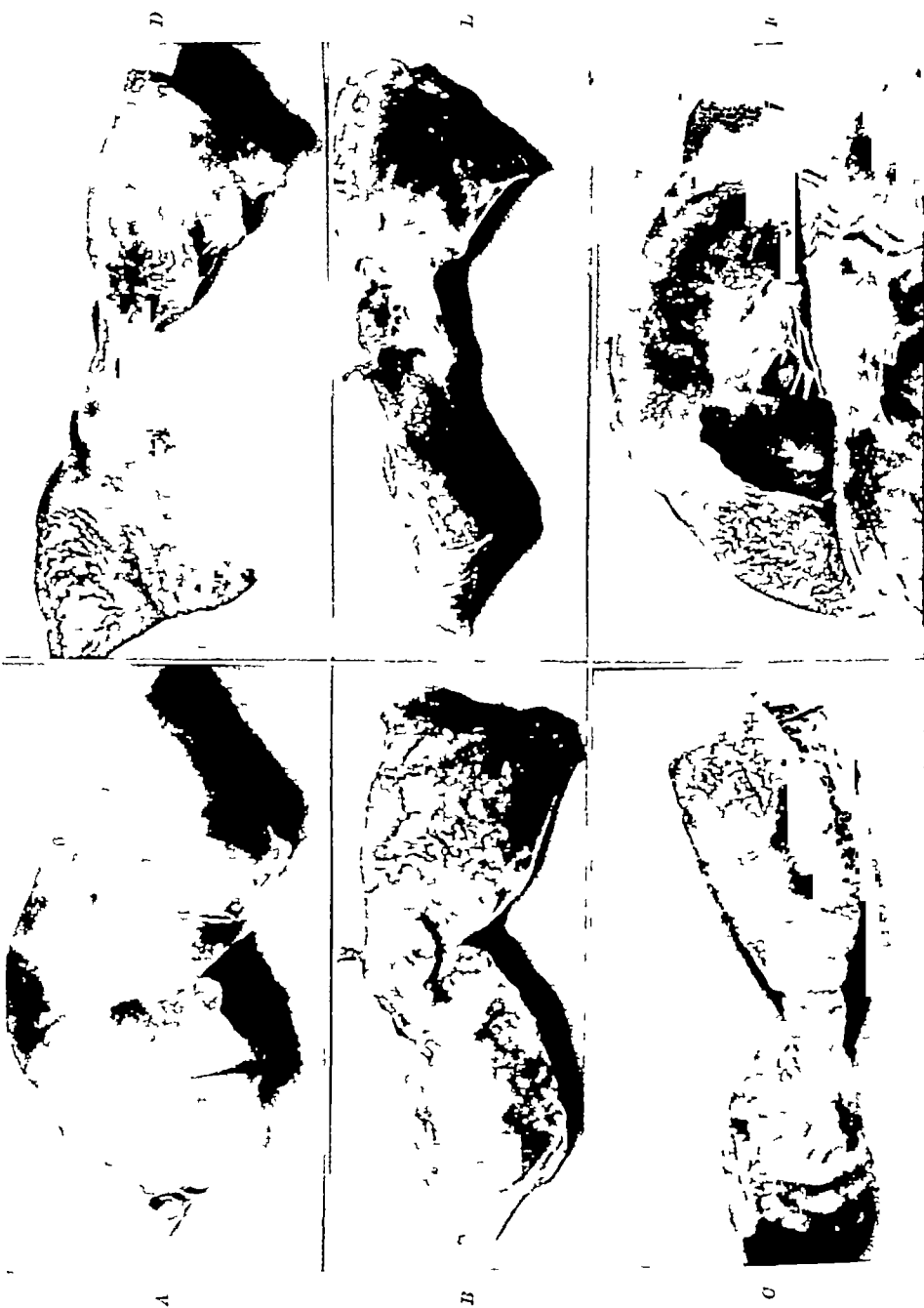


Fig. 1—Cross sectional view through midportion of scar at indicated interval from time of cardiectomy. Note variation in breadth and depth of scar. A, One week; B, two weeks; C, one month; D, two months; E, three months; F, twenty months.

was variable in width, and in a few instances involved the entire portion of the myocardium imprisoned by suture material. The necrotic muscle was rapidly invaded and replaced by granulation tissue apparently arising from adjacent viable myocardium (Fig 5). Complete replacement was usually present between two and three weeks. The more mature scar was characterized by an abrupt transition from relatively normal myocardium to dense, at times almost hyaline, cicatrix. The production of cartilage and bone was observed in one instance. Considerable reaction was present about the suture material, and, as was noted grossly, the scar usually extended two to five millimeters beyond the sutures.

CHANGE IN DEPTH OF SCAR FOLLOWING *INCISION* OF VENTRICULAR WALL

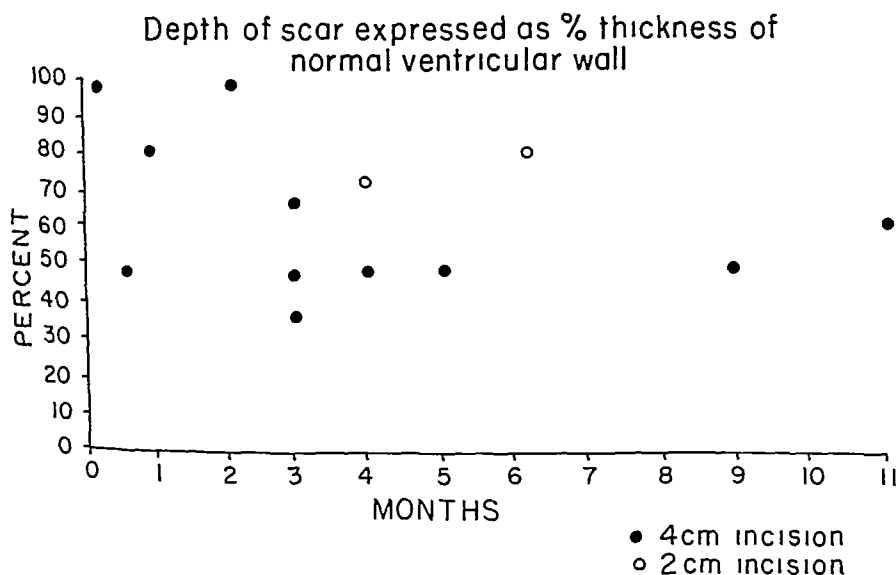


Fig 4

2 *Endocardial healing*—Within one week both the suture material and denuded endocardial surface were covered with a thin layer of fibrin. Rapid surfacing with a mesothelial or endothelial-like lining occurred which was usually complete at two weeks. Thereafter the endocardial scar remained smooth, with the suture material well embedded within the scar.

A limited number of observations over a period of eleven months were made on healing following placement of sutures without engagement of the endocardium. They would suggest that attenuation is the same or somewhat less than when through-and-through sutures are placed. Furthermore, broadening of scar was less conspicuous on the endocardial surface. These findings may be attributed to the failure of incorporation of the endocardium resulting in less tissue necrosis and to the foreign body reaction being limited to the myocardium.

3 *Thrombus formation and embolism*—There was no evidence of embolism in any of the dogs observed. In seventeen hearts examined between one and ten days postoperatively, only two instances of mural thrombus occurred. In one of these, a relatively large area of necrosis was present at the suture line.

4 *Epicardial healing*—The epicardial scar was greater in breadth than that on the endocardial surface, and was adherent to the pericardium. In a few animals the suture material seemed to be extruded toward the epicardial surface (Fig 3, *E*, three months).

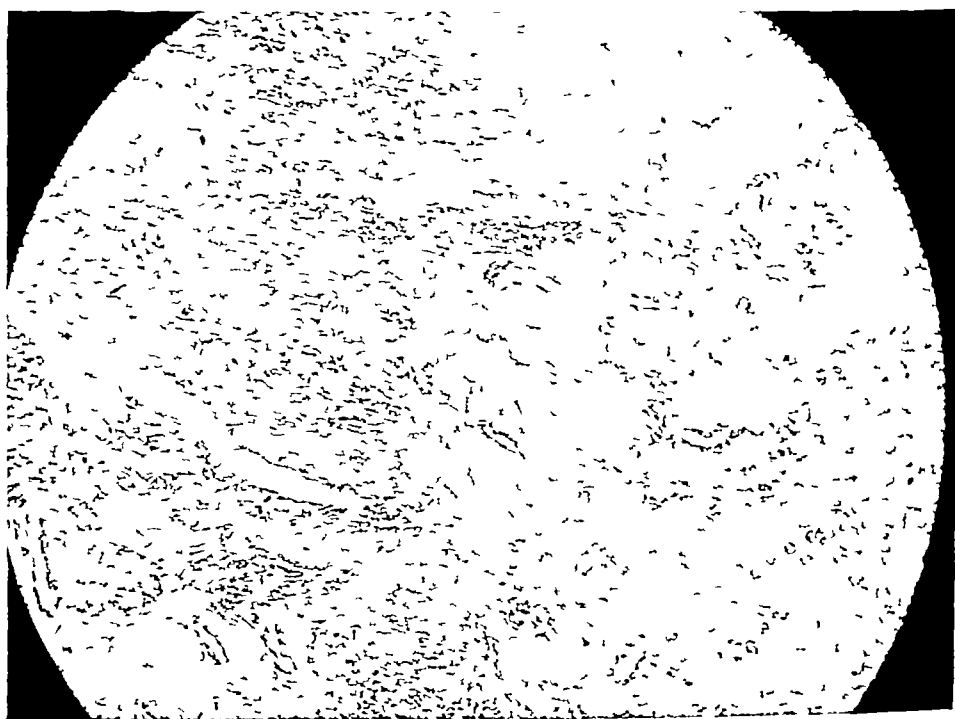


Fig 5—Healing at two weeks. Note replacement of dead muscle by granulation tissue.

5 *Wound dehiscence*—Rupture of the myocardium at the site of suture occurred in three of twenty-seven dogs surviving partial excision of the ventricular wall, and in three of eighteen dogs surviving linear incision for an over-all incidence of 13.3 per cent. This complication occurred early in the course of the experiments, and with greater experience was practically eliminated. In every instance in which rupture occurred there was necrosis of a portion of the myocardium contained within the suture line. Dehiscence occurred within one to seven days postoperatively.

6 In no instance did death occur from bleeding at the site of the suture line or from cardiac tamponade.

7 *Aneurysmal defects* did not develop, although in three animals (Figs. 1 and 2) the scars measured between one and two millimeters at the thinnest point. It is conceivable that if these animals had been permitted to survive a longer period of time aneurysmal dilatation might have occurred.

8 *Cardiac failure* was not observed.

9 *The electrocardiographic findings* were those of mild changes in the T waves similar to those observed by Fowler and associates⁴

10 *Adhesive pericarditis*—A significant number of animals were noted to have an adherent pericardium which snugly fit the epicardial surface. This was particularly adherent at the site of myocardial incision and damaged epicardium. Although in many animals the pericardium seemed to tightly encompass the heart, there was no evident increase in venous pressure, ascites, or peripheral edema. In those hearts with a "tight" pericardium, the heart was seen to bulge through the pericardium upon its incision. Although cartilage and bone formation were observed in myocardial scars, none was noted involving or arising in adherent pericardium. No relationship was observed between time and adherence of pericardium and essentially none between the type of experimental procedure and the degree of adherence.

DISCUSSION

These findings indicate that, despite a relatively large cardiac wound, repair is carried out in a manner similar to that observed in other tissues in which regenerative capacity is nil. Thus, the defect created is effectively bridged for functional purposes. Although on a theoretical basis sufficient attenuation of a large wound to produce rupture or an aneurysm would seem a real possibility, it was not observed experimentally. It is apparent that the degree of attenuation is dependent upon several factors. The relatively greater depth of scar observed after linear incision as compared to partial excision can probably be attributed to a nicer coaptation of wound margins so that the entire ventricular wall is in apposition. It is unlikely that any theoretical changes in inter-ventricular tension due to decrease in size or ventricular capacity would account for such differences. There was less attenuation in relatively short, two-centimeter, incisions than in those twice this length. Although in general the degree of attenuation remained fairly constant regardless of the thickness of the ventricular wall, those regions near the apex measured the thinnest in terms of millimeters, and it is in these regions that an aneurysmal dilatation would seem most likely to occur. Another factor must be the amount of muscle made ischemic by an encircling suture. If great enough the course of repair would be more analogous to that of a healing myocardial infarct.

Although it has been recommended that sutures be placed so they do not penetrate the endocardium,^{3, 10} this requires a nicety of placement that is seldom realized under the present circumstances. Furthermore, the sutures within the ventricular lumen are soon covered with a thin layer of fibrin and are no more dangerous as a source of emboli than the denuded cardiac muscle, although theoretically, with perfect wound coaptation, the endocardial surface would be intact. As has been the experience with intimal sutures within the aorta, the presence of a rapid blood flow seems to minimize the chance of a large thrombus being formed and subsequent embolization.

A limited number of observations suggest that additional cardiac load produced by an arteriovenous fistula, sufficient to result in mild cardiac hyper-

trophy, does not materially affect the course of healing as described. Increase in body weight—heart weight ratio was used as a criterion of hypertrophy.¹²

Wound dehiscence seems primarily to depend upon the amount of necrosis of cardiac muscle caused by the wound closure. With experience in the placement of sutures and the attainment of appropriate degrees of tension, this complication should be obviated.

SUMMARY

1. Repair of large cardiac wounds in the dog was characterized by production of a dense scar uniting the wound margins without apparently impairing cardiac function.

2. Attenuation of this scar occurred so that at one month it measured 50 per cent of the thickness of the adjacent normal ventricular wall and usually continued until the scar measured 30 per cent of this thickness. These changes occurred over a period of several months.

3. There were considerable variations in the breadth of the scar.

4. No evidence of aneurysm was observed in the scars under normal and under increased cardiac load.

5. Embolism was not observed following any of the procedures.

6. The strongest scar was obtained with the shortest incision through the thickest part of the myocardium in conjunction with maximum wound apposition by sutures causing minimal ischemia.

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HEMOPERICARDIUM AND CONSTRICTIVE PERICARDITIS

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THE development of the majority of cases of constrictive pericarditis is not well understood. Tuberculosis has been implicated in from 10 to 28 per cent of various collected series such as those reported by Beck¹ and Chambliss. The remainder have been ascribed to acute pyogenic infection, rheumatic fever, polyserositis, or unknown causes. The pathologic findings of those in the latter categories usually show nonspecific fibrosis with or without calcification. Frequently there is nothing to suggest tuberculosis or a specific etiologic agent. It is this group of patients we are concerned with in this publication.

The occurrence and subsequent operation of a patient with chronic hemopericardium and early constrictive pericarditis which developed following nonpenetrating minimal precordial trauma was recently observed by the authors. Relatively minor precordial trauma, followed by development of a chronic hemopericardium, was clearly established in the development of this case. The postulation of such a sequence of events operating in patients with nonspecific constrictive pericarditis is worthy of further study. The etiologic relationship between pericardial hemorrhage produced by trauma and late chronic constrictive pericarditis has perhaps been overlooked. Chronic pericardial effusion and pericardial hemorrhage may remain relatively asymptomatic for long periods of time until definite symptoms of constrictive pericarditis become apparent. This latent period of varied length has been mentioned in the few reported cases.

REVIEW OF LITERATURE

Interest in the etiology and development of constrictive pericarditis was first recorded by Beck in 1929³ and 1930,⁴ who investigated the results of Dakin's solution placed within the pericardial sac. At this time the possible end results by using this solution for irrigation in cases of purulent pericarditis were under investigation using dogs as experimental animals. A hemorrhagic pericarditis was produced by this solution which was followed by late constrictive pericarditis.

The possible role of hemorrhage into the pericardial sac and its relation to constrictive pericarditis was first suggested by Daniel and Puder⁵ in 1932. Autopsy findings in their case consisted of precipitated cholesterol within the pericardium. Hemorrhage, followed by precipitation of blood lipids and subsequent pericarditis, was offered to explain the pathogenesis. White⁶ mentions the possible role of traumatic hemopericardium in the etiology of

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²Presented at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery at Dallas, Texas, May 8-10, 1952.

adhesive pericarditis in the 1937 edition of his textbook. Extensive constrictive pericarditis was found at autopsy by Glenn⁷ in a patient who had suffered a crushing injury to the chest three years ante mortem. This author felt that traumatic hemopericardium was responsible in his case. Hemosiderin and lipids were found microscopically in a case of hemorrhagic pericarditis by Voldet.⁸ Recurrent hemorrhage was believed the etiologic factor in this presentation, and the author implicated either trauma or myocardial infarction as the source of bleeding. Pericardiectomy was performed by Dejon⁹ in a 19-year-old soldier for hemopericardium which followed nonpenetrating chest trauma. Syphilis was implicated in one of four cases of chronic hemorrhagic pericarditis by Froment and associates.¹⁰ One of three patients with chronic constrictive pericarditis reported by Barker and Johnston¹¹ required pericardiectomy three years following trauma to the chest cage. Ada and associates¹² report a somewhat similar situation, and present the microscopic findings. The patient had experienced three episodes of nonpenetrating chest trauma during the two years prior to pericardiectomy.

The most recently reported case was that of Overholt and associates.¹³ Bilateral fibrothoraces accompanied the chronic constrictive pericarditis. Decortication of all three sites resulted in improvement. Nonpenetrating trauma preceded surgical treatment by five years, and the symptoms resembling Pick's disease were well developed at that time. Sterile hemopericardium and hemothorax were thought to be the etiologic factors. This is the first recorded case studied pre- and postoperatively by cardiac catheterization and ventilatory function studies.

The aspiration treatment of pericardial tamponade following penetrating cardiac wounds has been reported by Elkin and Campbell¹⁴ and Ravitch.¹⁵ Development of the process under discussion in this situation would seem a real possibility. The question has been raised whether blood left within the pericardial sac would not eventually lead to the development of constrictive pericarditis. We did not observe blood to be clotted within the pericardial sac of the case which is presented, but that this may occur has been mentioned by Maguire and Gage.¹⁸ Well-developed chronic constrictive pericarditis was found in one of Maguire's patients who had been treated for a penetrating cardiac wound by aspiration six months previously.

Unfortunately, the type of patient most likely to incur this injury is not often available for follow-up study. Chronic constrictive pericarditis has not been observed by either Elkin¹⁶ or Ravitch¹⁷ in their cases treated by pericardial aspiration. That this train of events may occur more frequently than the literature indicates seems entirely possible.

Two cases of cholesterol pericarditis are to be found in the English literature. The most recent by Ada and associates¹⁹ was of unknown cause. A chronic constrictive pericarditis was found. The granulation tissue and scar contained cholesterol crystals. Unrecorded and unremembered precordial trauma producing hemopericardium must be considered as a possible etiologic agent.

In a collected series of 261 cases of nonpenetrating injury to the chest, Warburg²⁰ found evidence of pericardial hemorrhage in 23 per cent

Kerley²¹ feels that cases of calcific pericarditis may well be due to a traumatic hemopericardium rather than to specific infections such as tuberculosis

CASE REPORT

K. D., a 35 year old white truck driver, entered the University Hospitals on Jan. 4, 1951. Admission was prompted because of bleeding from a left bronchopleurocutaneous fistula which had persisted after drainage of a lung abscess at the age of 4 years. He was a well developed and well nourished man in no acute distress. He was afebrile, and the blood pressure was 120/85. A large bronchopleurocutaneous fistula overlying the left fifth rib was present. The remainder of the physical examination was essentially normal.

Bronchograms revealed bronchiectasis involving the lingular segment of the left upper lobe and the bronchial fistula leading into this pulmonary segment. On Jan. 10, 1951, a left upper lobectomy was performed. Convalescence was uneventful, and the patient was discharged in good condition on Jan. 21, 1951. An electrocardiogram was normal. Roentgenograms of the chest at the time of discharge showed a normal heart shadow and a well expanded left lower lobe.

The patient was readmitted on July 25, 1951. He had gained twenty one pounds, and had resumed work. No unusual symptoms were noted, but upon more thorough questioning it was discovered that he had noticed some shortness of breath and easy fatigue during the past few weeks.

A routine roentgenogram of the chest revealed a huge pericardial shadow. This was an entirely unsuspected finding, and it was only following further questioning that he recalled being struck by a baseball over the precordium six weeks prior to this admission. On July 27, 1951, the pericardial sac was tapped and 20 cc of old, dark blood was removed. Venous pressure readings were 170 mm of saline. On July 30, 1951, the pericardial tap was repeated, and at the same time cardiac catheterization studies were carried out in the cardio-

TABLE I. COMPARATIVE STUDIES OF PERICARDIAL FLUID AND PERIPHERAL VENOUS BLOOD

	PERICARDIAL FLUID			PERIPHERAL VENOUS BLOOD		
	T	A	G	T	A	G
Plasma proteins	660	432	228	735	463	272
Chlorides	680	mg	per cent	675	mg	per cent
Cholesterol	66	mg	per cent	108	mg	per cent
Cholesterol esters	36	mg	per cent			
Blood urea nitrogen	18	mg	per cent	18	mg	per cent
Creatinine	2.2	mg	per cent	1.6	mg	per cent
CO ₂ combining power	47	cc		54	cc	
Oxygen content	2.08	vol	per cent	16.36	vol	per cent
Van den Bergh	15	mg	per cent—1 min			
	15.6	mg	per cent—30 min			
	204	mg	per cent			
Hemoglobin with related heme pigments (normal 20 mg per cent)	8.1	Gm		15.3	Gm.	
Hemoglobin	22	per cent		52	per cent	
Hematocrit	2.62	million		5.23	million	
Erythrocyte count	5,500			7,800		
Leukocyte count	segs	6	per cent	segs	71	per cent
Differential count	lymphs	93	per cent	lymphs	26	per cent
	monos	1	per cent	monos	2	per cent
				cosinophils	1	per cent

vascular laboratory * Three-hundred eighty cubic centimeters of dark blood were withdrawn, and two hundred fifty cubic centimeters of air injected. Withdrawal of the fluid caused simultaneous decline of elevated intrapericardial and right intra atrial pressures toward normal. The fixed cardiac output revealed a significant degree of tamponade or constriction. Comparative studies of pericardial fluid and peripheral venous blood are shown in Table I.

On Aug 3, 1951, a partial pericardiectomy was performed. Most of the pericardium overlying the left ventricle and right ventricle was excised. About 500 cc of old, nonclotted blood were removed from the pericardial space. The pericardium was noted to be four to six times its normal thickness. There was extensive organizing granulation tissue over the epicardium and pericardium. No significant bleeding points were found within the pericardium at operation. An electrocardiogram showed a decrease in the QRS and T waves. His postoperative course was uneventful, and he was discharged on Aug 12, 1951.

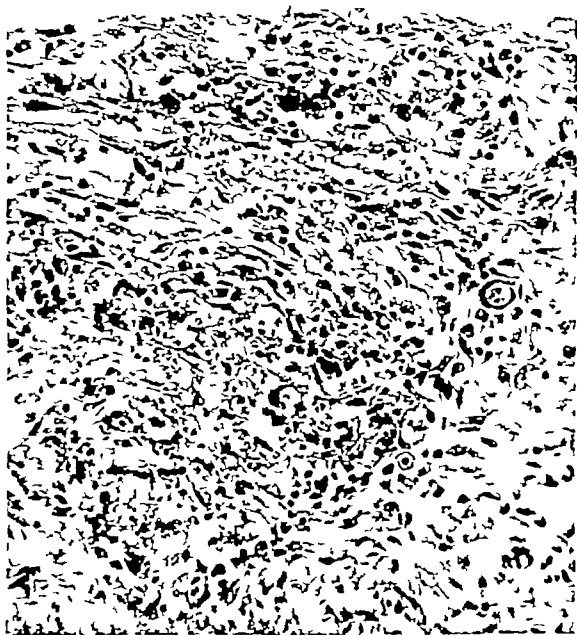


Fig 1—Photomicrograph of excised pericardium showing organizing granulation tissue of reported patient with chronic hemopericardium.

Microscopically the sections show extensive nonspecific granulation tissue and scar. The strikingly thickened pericardium showed scarring in various forms of organization from fibrin most superficially to relatively avascular laminated collagen in the deeper layers. There was chronic inflammation and foreign body giant cell reaction present. Considerable vascularity was seen throughout, most apparent in the areas of organizing granulation tissue. Many phagocytes containing blood pigment were noted (see Fig 1). The pericardial blood was inoculated into a guinea pig and reported as negative for tuberculosis. A bacterial culture proved it to be sterile.

The patient was readmitted on Aug 24, 1951. His vital capacity at this time was 2.8 liters. He had been working and felt much better than he had prior to the last admission. The venous pressure was 85 mm. of saline and the circulation time, arm to tongue, was 18 seconds using Decholin. A roentgenogram of the chest revealed a heart of normal size. A roentgenkymogram showed normal cardiac movements. An electrocardiogram showed only inverted T waves. Cardiac catheterization was performed and revealed essentially normal resting values (see Table II). He was discharged on Aug 28, 1951.

*These findings will be reported in detail by Ames and associates *American Journal of Medicine* (in press).

The last admission occurred on Feb 26, 1952. A roentgenogram of the chest revealed again a normal cardiac silhouette (see Fig 2). Resting values during cardiac catheterization at this time were essentially normal. He did, however, have a fixed cardiac output under exercise. This was interpreted as showing some evidence of limited cardiac expansion. The

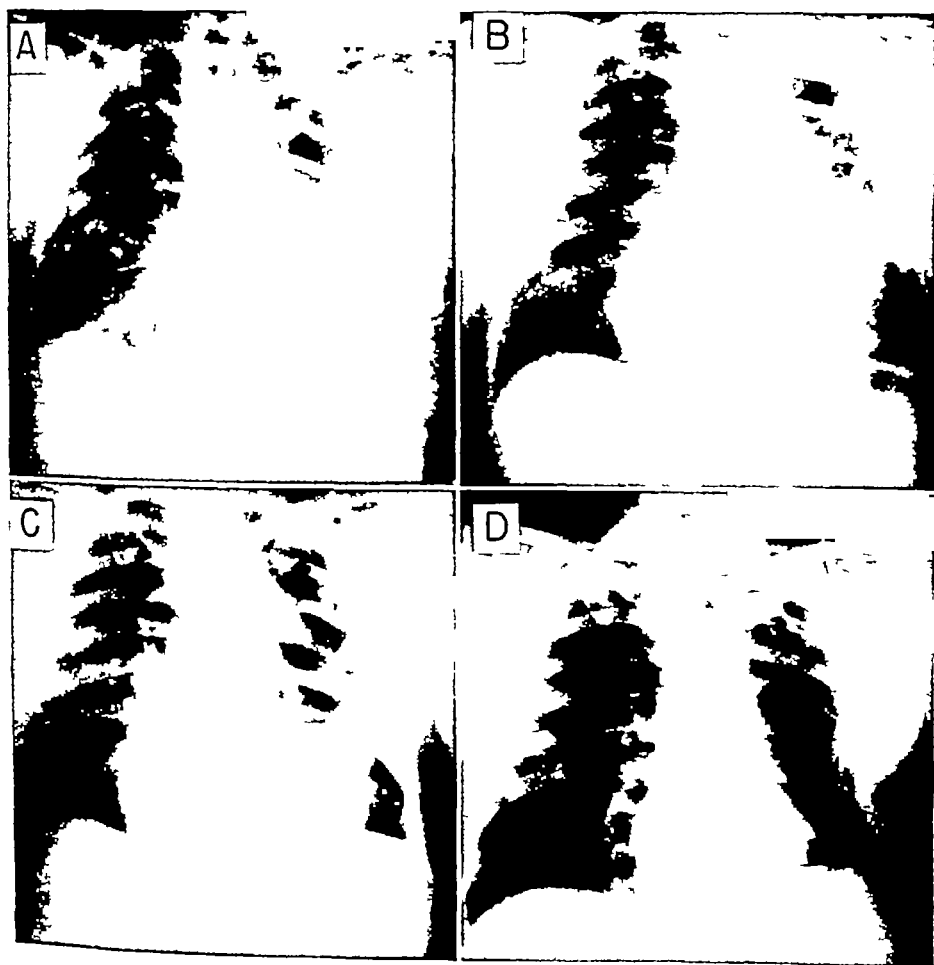


Fig 2.—Roentgenograms of chest of patient with chronic hemopericardium. A Jan 21 1951 normal configuration of heart following left upper lobectomy. B July 23 1951 massive pericardial effusion at time of first readmission. C July 30 1951 after withdrawal of 380 c.c. of pericardial fluid and injection of air. D Feb 26 1952 normal cardiac configuration after pericardiectomy at time of final admission.

patient is being observed at the present time for the development of further symptoms. It is possible that he will require excision of the thickened epicardium which was a poorly defined layer at the time of the pericardiectomy.

EXPERIMENTAL DATA

The poor absorptive powers of the rabbit pericardium were studied by Drinker and human pericardium by Stewart²³. They found that electrolytes but not colloid particles were taken up by the pericardium. The rabbit peri-

TABLE II CARDIAC CATHETERIZATION DATA

PRESSURES (MM HG)	NORMAL	PREOPERATIVE	3 WEEKS POSTOPERATIVE
Right atrium	8/0	20/15	10/2
Right ventricle	30/0	32/12	21/0
Pulmonary artery	30/15	31/16	26/15
Pulmonary capillary	6	12	5
Cardiac output at rest (liters per minute)	6.0	6.4	6.75

cardium was found to contain very few lymphatic channels which accounted for the scanty colloid removal. Electrolytic solutions were easily removed through vascular channels.

The well-defined sequence of events, namely, hemopericardium produced by nonpenetrating precordial trauma, followed in seven weeks by the development of early constrictive pericarditis in the case presented, prompted us to make the following investigation. An attempt was made to produce chronic constrictive pericarditis by injecting the lipid fraction of dog's blood into the pericardial sac. Upon suggestion by Dr. N. A. Womack the lipid portion was utilized because it was thought that it might be the most likely fraction to produce inflammatory response. Another series of animals was maintained simultaneously by injecting autogenous blood into the pericardial sac.

Fourteen mongrel kennel dogs were utilized. The injections were made under intravenous Nembutal anesthesia using controlled endotracheal oxygen ventilation. The animals varied in size between nine and fourteen kilograms. A left thoracotomy was carried out, and the injections made through a needle in the pericardial sac. The needle wound was ligated to prevent leakage of the injected material. The series of animals in which blood was used served as their own donors, the blood being aspirated from the auricle or ventricle and injected directly into the pericardial sac. The largest dogs received 100 cc of blood, and the smallest received 50 cc. More than this amount of injected blood frequently precipitated acute tamponade, and required partial removal of the injected material.

The series of animals receiving lipid extract were operated upon under similar conditions. They received the lipid fraction of 100 ml of dog blood in 25 ml of phosphate buffer suspension having a pH of 7. This suspension was injected in a like manner. The lipid fraction was extracted from pooled dog's blood by the following method. The blood was collected in 500 ml of extracting fluid containing 1:1:1 ratio of acetone, alcohol, and ether. The resultant suspension was filtered through a Buchner funnel and washed with several portions of extracting fluid. The filtrates were pooled and evaporated. The lipid fraction was prepared for injection by placing 600 mg of the evaporated material in 25 ml of phosphate buffer, emulsifying it in a Waring blender. The material was then sterilized in rubber stoppered bottles at 15 lb pressure for fifteen minutes. Plasma determinations on whole donor dog blood showed cholesterol 130 mg per cent and proteins 6 to 8 Gm. An analysis of the lipid fraction extracted from pooled whole dog's blood yielded the

Following 600 mg lipid fraction contained cholesterol (Liebermann-Burchard method) 52 mg or 87 per cent, phospholipids (Gibson's method) 490 mg or 81.7 per cent, and the remainder, 58 mg or 9.6 per cent of other lipid material.

Two dogs each of the blood and lipid series were sacrificed at intervals of one, three, and six months. Two dogs injected with a suspension of crystalline cholesterol in saline were sacrificed at six and ten weeks each.

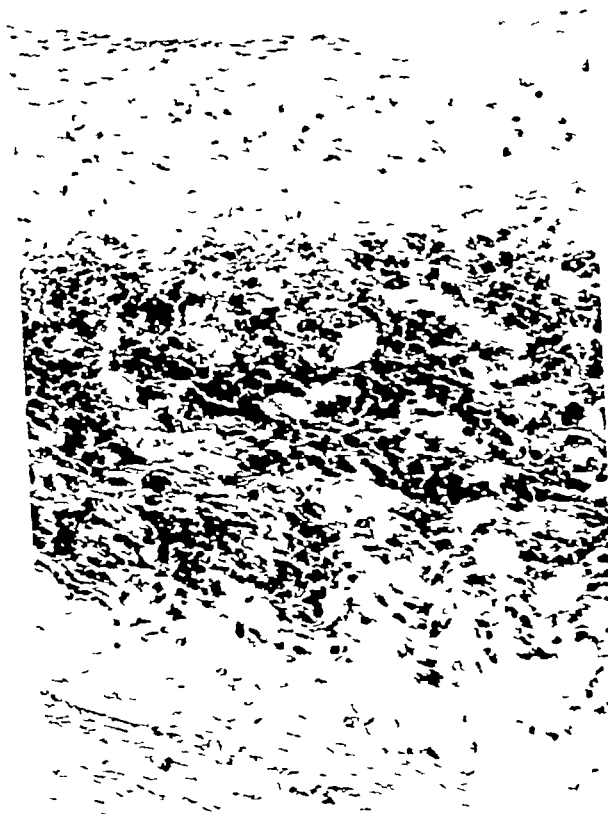


Fig 3—Photomicrograph of epicardium of a dog sacrificed one month after injection of 100 c.c. of autogenous blood. Scarring inflammatory response and pigment-laden phagocytes are seen (iron stain).

EXPERIMENTAL RESULTS

All animals of both series showed changes in the pericardium and epicardium. The amount of scar tissue response varied from rather minimal changes in some of the dogs with hemopericardium to far advanced hemorrhagic constrictive pericarditis in one dog of the lipid series. The pericardial tissue of all animals was examined microscopically using hematoxylin-eosin, iron, and special fat stains. None of the dogs who had autogenous blood injected into the pericardial sac developed advanced changes or constrictive

pericarditis. These animals showed patchy pericardial and epicardial scarring without formation of a constrictive envelope. Adhesions between the pericardial and epicardial layers were not a prominent feature. None of the

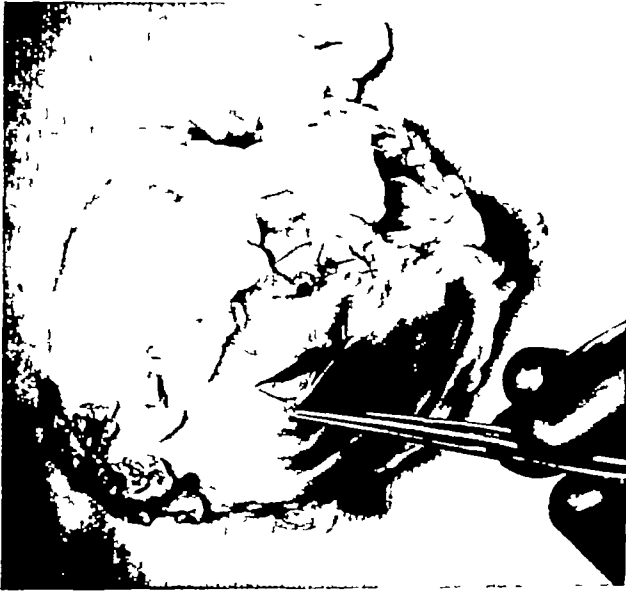


Fig. 4—Photograph showing heart of a dog injected with blood lipid fraction sacrificed at three months. Adhesive pericarditis is demonstrated.



Fig. 5—Photograph showing the dog heart seven weeks after injection of blood lipid fraction. Extensive hemorrhagic constrictive pericarditis is shown.

injected blood was found in any of the dogs at the time of autopsy, and none of the pericardial findings were more marked in the dogs sacrificed at longer intervals

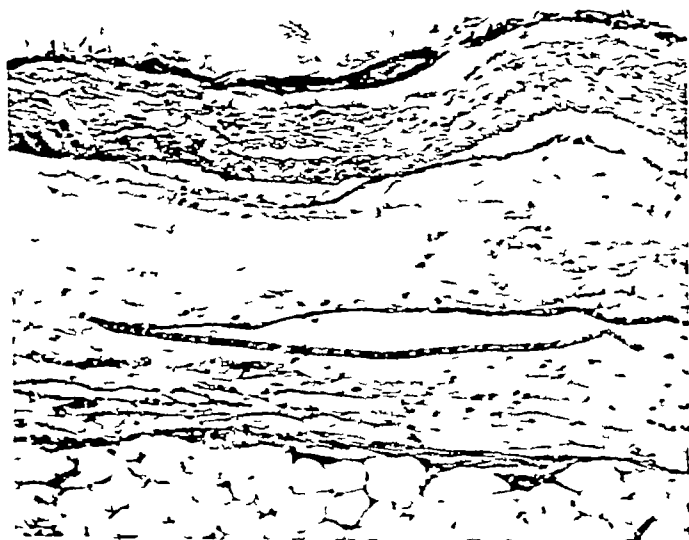


Fig 6—Photomicrograph showing adhesive pericarditis in a dog injected with lipid suspension three months before sacrifice.

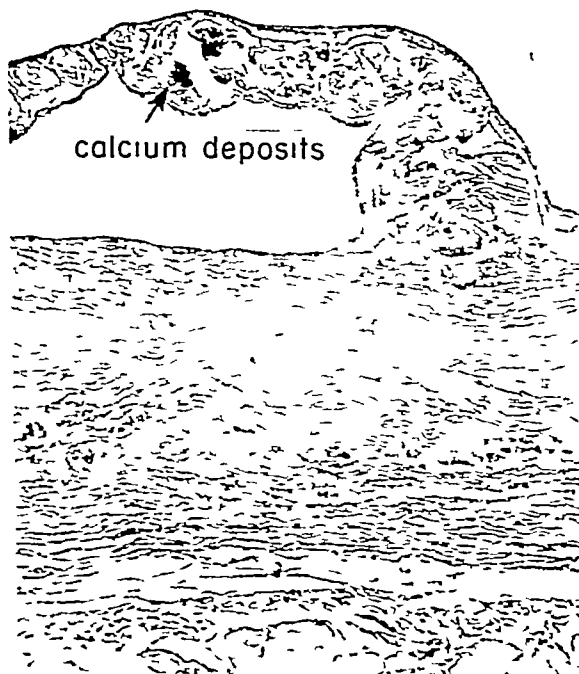


Fig 7—Photomicrograph showing organizing granulation tissue containing calcium deposits and the scarred pericardium. The animal died seven weeks after injection of blood lipid.

Microscopically, the areas of patchy pericardial thickening showed inflammatory reaction and scarring with dense collection of phagocytes filled with blood pigment (see Fig 3)

Animals who had the blood lipid fractions injected all showed more advanced changes. Those changes consisted in pericardial and epicardial thickening, patchy areas of granulation tissue, and adhesions. The changes were more advanced in the animals which were sacrificed at longer intervals (see Fig 4). No evidence of pericardial effusion or remnants of the injected lipid suspension were found free within the pericardial sac.



Fig 8—Photomicrograph demonstrating phagocytized lipid material within the scarred epicardium. This is the same animal as demonstrated in Fig 7 (Sudan III stain.)

One of the animals died at seven weeks with advanced findings resembling Pick's disease due to constrictive pericarditis. Grossly the animal had generalized edema, ascites, and bilateral pleural effusions. The heart was small, constricted, and surrounded by a densely thickened pericardial and epicardial layer. A small amount of hemorrhagic sterile fluid was present in the pericardial space (see Fig 5).

Microscopically the presence of adhesive pericarditis with slight inflammatory response was found in some of the less-advanced specimens (Fig 6). In the animal which died of the constrictive pericarditis, extensive inflamma

tory response and scarring were present. Considerable amounts of organizing fibrin, some of which contained early calcium deposits were found (Fig 7). Fat stains (Sudan III) demonstrated phagocytized lipid material within the extremely scarred epicardium and pericardium (Fig 8).

The two dogs which received crystalline cholesterol suspension injections into the pericardial sacs, sacrificed at six and ten weeks each, showed essentially the same findings as the majority of the dogs receiving the lipid extract. Well-developed constrictive pericarditis was not produced in those animals.

SUMMARY

A review of the literature has revealed several cases which show the sequence of traumatic hemopericardium followed at varying intervals by signs and symptoms of constrictive pericarditis. The patient reported in this presentation adds additional evidence to this line of thought.

A series of dogs received an intrapericardial injection of autogenous dog's blood and a second series injections of the lipid fraction of pooled dog's blood. Only one animal in the latter series demonstrated well-developed constrictive pericarditis. The remainder of the dogs in the lipid series showed lesser changes. The animals in the blood series showed the least changes. These experimental findings are interpreted as supportive evidence that in some instances constrictive pericarditis may be the end result of intrapericardial hemorrhage. It would also appear that the lipid fraction of blood is probably the significant component in the production of pericardial reaction.

This problem is not considered closed until similar experiments have been carried out using repeated injections of blood and lipid fractions of blood to simulate more long-standing hemopericardium.

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DISCUSSION

DR C THOMAS READ, Phoenix, Ariz.—Early in our studies of the enzymes in New York, we were asked to see a patient who was practically moribund with Friedlander's pneumonia on the left side. Apparently an empyema of the pericardium had formed and we were asked to investigate this situation relative to the use of the enzymes. We thought this offered an opportunity to study an unusual situation so a small polyethylene catheter was inserted into the pericardial sac by way of a large needle of about 12 to 13 gauge. When the catheter was in the pericardial sac, the needle was removed. The catheter was then attached by way of another smaller needle (cannulated within the catheter) to a three-way stopcock. The catheter was left in situ for a period of thirty-six hours, and the three-way stopcock was put there in order that, if evidence of a tamponade existed, relief of pressure could be carried out. The original specimen of material aspirated was frankly purulent and thick. Upon about three treatments with the enzyme material, an almost serous fluid was the end result. In this situation, whether it was the treatment of the tamponade, or the purulent pericarditis, we do not know, the patient made a recovery so far as the pericardium was concerned. I offer this method as another way in which hemopericardium might be managed.

DR MARK M. RAVITCH, Baltimore, Md.—As Dr Ehrenhaft stated, Dr Elkin, Dr Bigger, and others have been interested in pericardial aspiration of acute traumatic tamponade, and Dr Blalock and I have now a series, with the house staff, of 18 or 19 cases of acute traumatic pericardial tamponade, treated with aspiration. As Dr Ehrenhaft indicated, the citizens who are the subjects of such injuries have a certain self interest in remaining unavailable, and we have been able to trace only four of our patients who have been intensively studied by Dr Robert Mason. None of them had aneurysms, none had constrictive pericarditis, and none had electrocardiographic abnormalities. We have had the opportunity to explore the pericardium in only one patient after what appeared to be a successful aspiration. This was a man who was shot through the heart. His electrocardiogram, taken almost as soon as he arrived at the hospital, was diagnosed as showing a block of the anterior descending coronary artery. He did have pericardial tamponade. All his indices were reversed by the aspiration treatment. He died in cardiac failure within a couple of hours. Autopsy showed the injury to the coronary artery and only two or three cubic centimeters of blood at the bottom of the pericardium.

We have had in Dr Blalock's series of constrictive pericarditis, one patient who had a very severe crushing injury to the sternum years before, who appeared to have a typical chronic constrictive pericarditis. However, we have a clinical observation which leads me to ask Dr Ehrenhaft a question about the case which he is reporting. This was a Negro who was stabbed through the left chest, had a typical pericardial tamponade which was dramatically relieved by the aspiration of blood, who was admitted to the hospital and stayed

there for several days, when the tamponade returned slowly. The pericardium was aspirated, re-aspirated, and aspirated again, and it soon became apparent that this was bloody fluid and not blood, that he was not leaking from the heart, and a more careful check showed that he had active apical tuberculosis on the left and, in fact, he turned out to have a tuberculous pericarditis. That man may well go on to develop a chronic constrictive pericarditis. We presume that the wounding instrument either went through the lung or, possibly, through a tuberculous mediastinal lymph node, and it was interesting to me that Dr Ehrenhaft's patient did have some sort of pulmonary difficulty.

DR ADRIAN LAMBERT, Jr, New York.—I recently had occasion to operate on a patient with chronic hemopericardium of approximately three years' duration. The cause of this was unknown. He had had for the last year symptoms of cardiac tamponade that were relieved by aspiration, which had become ineffective for the last three weeks, just before our exploration. At the time of operation, in spite of vigorous supportive treatment, he came to the operating table with marked anasarca. On opening the pericardium approximately 500 cc of chronic fibrinous bloody sludge was removed with old blood. As to the etiology of the hemopericardium, the cause is still unknown.

It was felt, because of the anasarca, that the patient preoperatively was suffering from constriction of the inferior vena cava as well as the cardiac tamponade, and at operation this was borne out. Over the visceral pericardium there was a plaque that was approximately 4 millimeters in thickness. Removal of this in one area was attempted but the heart muscle was so thin and edematous that the heart itself was perforated. This was controlled by resuturing the plaque, after which, as the patient suffered no untoward effects, the inferior vena cava was freed of its surrounding membrane. Following this the heart went into marked dilatation, in spite of the fact that the plaque was still present over its musculature. Arrest of the heart followed, which was relieved by manual massage so that permanent dilatation did not result. In retrospect, therefore, it was felt that the fact that we had not removed the plaque had actually saved the patient's life. This brings up the point whether, in these cases of constrictive pericarditis associated with tamponade, it might not be better, if there is constriction of the inferior vena cava, to free that first so as to avoid dilatation that cannot be controlled, reserving removal of the visceral plaque for a later date if constriction is not completely relieved.

DR ALEXANDER ADA, New York.—In March, 1948, I had the opportunity to observe a patient who had a pericardial effusion. In the previous five months he had received three distinct contusions of the anterior chest wall. The notable findings on examination were dyspnea, cyanosis, distention of the neck veins, and an enlarged heart. The venous pressure was 290 mm. of saline. Pericardial tap performed in December, 1947, produced 400 cc of blood. The diagnosis of hemopericardium was made and, due to the persistence of the latter and the possibility of a neoplastic process or a vascular lesion, pericardiectomy was advised.

At operation the pericardium was found to be bulging, densely thickened, and lined throughout by fine vascular granulations which bled easily on contact. Large plaques of fibrin were present in the pericardial sac, and there were adhesions between the visceral and parietal pericardium. I felt that the pathologic process present would eventually lead to a constrictive pericarditis, therefore a resection of the anterior two thirds of the pericardium was performed. This also facilitated drainage of the pericardium into the mediastinum. The pathologic diagnosis was "chronic fibrous pericarditis with old and recent hemorrhage."

This is the first time, to our knowledge, that pericardiectomy has been performed for this particular lesion. Due to the increasing incidence of trauma to the chest, hemopericardium will occur more frequently and may escape diagnosis. Unquestionably, in a large number of these cases, the blood is absorbed without any apparent residual damage. On the other hand, untreated chronic hemopericardium will eventually lead to constrictive pericarditis.

Dr Ehrenhaft and Dr Taber are to be commended for their investigation of this particular problem and for bringing to our attention another etiologic factor in the production of pericardial constriction

DR EHRENHAFT (Closing) —We have not had occasion to try clinically or experimentally the treatment with enzymes, but I think it should be tried. If one reviews the literature one finds several cases of traumatic hemopericardium and constrictive pericarditis, and one is impressed that there are sometimes years between the time of injury, if it can be elicited from the history, and the evidence of constrictive pericarditis.

In answer to Dr Ravitch's discussion, I think that the injury in this patient and the hemopericardium are definitely related to each other, and not to be bronchopleural cutaneous fistula following drainage of a lung abscess at the age of 4 years.

I feel as Dr Ada does that in these patients pericardiectomy should always be done. I did not want to bring up the question of aspiration treatment versus pericardiotomy.

RUPTURE OF THE ESOPHAGUS

ROBERT L. ANDERSON, M.D. (BY INVITATION)

TULSA, OKLA

SPONTANEOUS rupture of the esophagus was first described in 1724 by Boerhaave²¹. From that time up to December, 1950, there were described in world literature ninety-two cases⁴⁰. Eighteen of these individuals survived.^{3, 4, 7, 10, 17, 18, 21, 22, 24, 27, 31, 34, 41} In 1951 there were, to my knowledge, twelve additional cases reported,^{5, 14, 25, 37, 40, 41, 44} eight of whom survived. It is the purpose of this paper to report four additional cases and to discuss problems associated with the etiology, diagnosis, and treatment of this condition.

In 1944 Graham²¹ described two cases that survived following surgical drainage of the pleural cavity. The first case that survived following thoracotomy and repair was reported in detail by Barrett³ in 1946. The first clinical diagnosis made before death is credited to Meyer in 1846²⁴. Even though this condition has been recognized for over two hundred years, the successful treatment has occurred largely in the past five years.

Relatively little has been written on this subject, and even now many physicians are unaware of its existence. Articles and textbooks reviewing the differential diagnosis of acute surgical emergencies often fail to mention rupture of the esophagus. Rupture of the esophagus probably occurs more often than is suspected. This condition should, therefore, be the subject of continued scrutiny.

CASE REPORTS

CASE 1—H. W., a 51 year old white man, was admitted to St. John's Hospital, Tulsa, Okla., at 9:40 P.M. on Nov. 25, 1950. The patient stated that he noted, while eating dinner at about 7:15 P.M., that he had a sensation of something "backing up into his throat." He took several swallows of water, in an attempt to wash the bolus of food down, with no effect. He felt a substernal pain that followed the last swallow of water. This was followed by a severe midepigastriac pain which lasted for several minutes and was then partially relieved. In about twenty minutes the pain recurred and was extremely severe. It had persisted from this time until the time of the examination. There was no retching or vomiting.

Past history revealed symptoms of peptic ulcer for which he had been under treatment. He had failed to continue himself to his ulcer regime. He was known to eat poorly, and consumed rather large quantities of alcoholic beverages. Roentgenograms in May of 1950 showed an active peptic ulcer.

Physical examination revealed a well developed, well nourished man. He was lying on his right side with his knees drawn up. He was not in shock. He had previously received 14 grains of morphine sulfate. His blood pressure was 128/78, pulse 112, respirations 28, and temperature 100.2° F.

Examination at this time was essentially negative except for the following findings:
1. as marked rigidity in the midepigastriac region with mild tenderness. There was

1. 1 at the Thirty Second Annual Meeting of the American Association for Thoracic Surgery at Dallas, Texas, May 9-10, 1952.

slight tenderness throughout the remainder of the abdomen. The lungs were negative to auscultation and percussion. There was no subcutaneous or mediastinal emphysema noted. Roentgenographic examination at this time revealed no air beneath the diaphragm and the chest was clear. No mediastinal emphysema was noted. A diagnosis of possible perforated ulcer was made, and a Levine tube was inserted and constant suction applied. Intravenous fluids and penicillin were given.

On November 26, the red blood count was 4.6 million, with 14.3 Gm. of hemoglobin. The white blood count was 22,300 with 94 per cent polymorphonuclear leukocytes. The serum amylase was 87 mg per cent at 10 A.M. and 122 mg per cent that evening. Urinalysis was normal.

On November 27, a roentgenogram of the chest revealed a pleural effusion on the right. Aspiration revealed a bloody, turbid fluid. It contained 18,400 red blood cells, 14,300 white blood cells, and was negative for free or combined hydrochloric acid. A diagnosis of rupture of the esophagus was made.

On November 28, a barium swallow was carried out which showed a defect in the esophagus in its lower third. Surrounding this was a mediastinal abscess, with free air in the mediastinum. Subcutaneous emphysema was felt in the left supraclavicular area. Aspirated pleural fluid was positive for hydrochloric acid. The patient's condition remained the same, although he complained of increased substernal pain, and pain over the posterior portion of the right chest.

On November 29, the patient was taken to surgery and a posterolateral thoracotomy incision was made over the ninth rib on the right side. The thoracic cavity was entered, and a good exposure to the lung and esophagus was obtained. The thoracic cavity was partially filled with sanguino-purulent fluid. The fluid was evacuated, the lower and middle lobes of the right lung were decorticated and re-expanded. However, after some difficulty a longitudinal slit, approximately $1\frac{1}{2}$ cm in length, was found on the anterior surface of the esophagus lying approximately two inches above the diaphragm. This perforation was closed in two layers, using black silk interrupted sutures. The thoracic cavity was thoroughly cleansed, and penicillin and dihydrostreptomycin were instilled into the thoracic cavity. The lung was re-expanded. The incision was closed with interrupted chromic catgut. Two large catheters, one posteriorly and one anteriorly, were left in place to facilitate drainage of the pleural space and mediastinum.

The individual had a somewhat stormy convalescence. The stomach was kept decompressed by means of a Levine tube and the patient was fed parenterally, receiving blood, plasma, protein hydrolysates, and glucose in Hartman's solution.

The total proteins on Dec. 12, 1950, were 5.12 Gm. per cent, with the albumin 2.49 Gm. per cent and globulin 3.23 Gm. per cent. Although subsequently the protein rose, the reversal remained.

Tube feedings were started on the seventh postoperative day, and gradually increased daily. Because of distention and nausea, the Levine tube was left in until the fourteenth postoperative day. Because of loculated effusion and clinical evidence of a localized mediastinitis, the intrapleural catheters were not removed until the fifteenth postoperative day.

He continued to improve, and was given diet by mouth on December 12. The patient was discharged on an ulcer diet on Dec. 24, 1950, which was twenty-five days following the operation. He has remained well up to the present time, and works every day. He is still on a restricted diet because of his ulcer history. He has a normally functioning esophagus, and barium swallow reveals no deformity.

CASE 2—V. McC., a 48-year-old white man, was admitted to St. John's Hospital, Tulsa, Okla., on March 6, 1950. At about 3 A.M. on March 6, 1950, this patient had a sudden onset of severe epigastric pain preceded by severe vomiting. He gave a previous history of having had digestive disturbances for which he frequently took soda. He had not been eating or drinking heavily before the onset of this episode, and gave no history of having been a heavy consumer of alcoholic beverages. At 6:30 A.M. the patient took a small drink of

water which was followed by a severe pain similar to the previous one. The patient was admitted to the hospital at 9 A.M., at which time his temperature was 100.4° F, pulse 124, and respirations 24.

Physical examination revealed the patient in severe pain. He was warm and moist, and had no symptoms of shock. Respirations were rapid and shallow. There was a tachycardia without thrills, murmurs, or irregularity. The lungs showed a slightly diminished tactile fremitus in both lower lung fields posteriorly, together with tubular breathing. No evidence of free fluid in the chest was found at this time. The abdomen was rigid with generalized tenderness. The peristalsis was diminished.

His blood count upon admission was 5.2 million red cells with 15.6 Gm. of hemoglobin, 17,650 white blood cells with 95 per cent neutrophils. The urine was normal. Roentgenogram upon admission showed no air beneath the diaphragm. Roentgenogram of the chest at this time showed evidence of fluid in the left thoracic cavity.

By 3 P.M. the epigastric pain had progressed into the anterior chest and was causing dyspnea with cyanosis. Temperature had risen to 101.8° F, the pulse was 124, and respirations 44. At 9 P.M. there was evidence of subcutaneous emphysema noted over the supraclavicular areas, face, neck and anterior chest wall.

The patient was given morphine, oxygen, penicillin, intravenous fluids and aureomycin.

On March 8 the patient's condition was still precarious, and consultation was sought. The consultant made a diagnosis of spontaneous rupture of the esophagus. The only treatment advised was a thoracentesis. The patient was taken to surgery, and a catheter was introduced into the thoracic cavity on the left and attached to an underwater drainage bottle. The Levine tube was used to give the patient tube feedings.

On March 9 the pleural fluid was tested for hydrochloric acid and proved negative. Roentgenogram still showed massive effusion in the left chest.

On March 14 the blood count showed 4.85 million red blood cells and 12,900 white blood cells, with 85 per cent polymorphonuclear leukocytes. The blood amylase was normal. The total protein was 5.77, with the albumin being 2.81 and globulin 2.96 Gm per cent. The chlorides and CO_2 combining power were normal. The left chest showed massive effusion.

On March 18 bismuth introduced by mouth was found by roentgenogram to be in the left thoracic cavity. Left pleural effusion was still present.

On March 22 the patient was discharged with a diagnosis of ruptured esophagus and empyema.

On March 24 thoracotomy was carried out in Dallas, Texas, by Dr. Robert R. Shaw. A perforation of the esophagus was found immediately above the diaphragm. This was repaired, the lung was decorticated, and the empyema cavity drained. Recovery was uneventful, and the patient was discharged in twelve days.

The patient has done well since, and is now completely well.

CASE 3—S. F., a 59-year-old white man, was admitted to the service of Dr. A. Ray Wiley, Hillcrest Hospital, Tulsa, Okla., on Oct. 12, 1950. Two hours previous to the time this patient entered the emergency room, he was sitting in a cafe, eating. He noticed, shortly after eating, that there was swelling of his eyelids and that his face was puffed up. This phenomenon continued and, after a short while, was associated with a severe cramping abdominal pain. He became progressively more short of breath. At the time he was seen in the emergency room he was in shock. He was complaining of severe cramping abdominal pain and shortness of breath. There had been no vomiting at any time.

His past history revealed he had had some difficulty in eating and swallowing for the past three weeks. There was also a history suggestive of peptic ulcer in the past. He was known to be a chronic alcoholic.

Physical examination revealed a well-developed, well-nourished man in shock and severe pain. He was cyanotic. There was subcutaneous emphysema around the eyes and over the face, neck, and the upper and anterior portion of the chest wall. There were crackles in both chest fields throughout both lung fields; however, breath sounds were heard but the sounds were diminished somewhat on the right side. The respiratory rate

was 40. He had an irregular pulse of 108. The heart showed no thrills and there were no murmurs noted. His blood pressure was 132/90. The abdomen was rigid and tender throughout.

Laboratory data showed a red blood count of ± 2 million, white blood count of 10,200, with 79 per cent polymorphonuclear leukocytes. The urine was not abnormal. Roentgenograms taken of chest and abdomen on this date reveal the presence of subcutaneous emphysema and mediastinal emphysema. There was no evidence of pneumothorax, atelectasis or fluid in the pleural cavity at this time. A diagnosis was made of ruptured viscus, probably esophagus.

The patient was given oxygen, morphine, and antibiotics. A Levine tube was introduced into the stomach and constant suction established. He was given blood and fluid intravenously. Conservative treatment was carried out over the next nine days. The patient received intravenous fluids, blood, stimulants, and intramuscular injections for pain.

On Oct. 14, 1950, fluid was seen by roentgenographic examination in both bases. There was more fluid on the right side and honeycombing of the lung occurred suggesting pulmonary edema, possibly on the basis of cardiac decompensation. Fluid was removed from both right and left thoracic cavities by thoracentesis. Further roentgenographic examinations of Oct. 20, 1950, showed the subcutaneous emphysema gone as was most of the pleural fluid. Some fluid remained in the left base. By barium swallow a diagnosis of hiatus hernia was made. No esophageal perforation was demonstrated.

The white blood count rose to 38,000 with 93 per cent polymorphonuclear leukocytes. The total serum proteins were 5.5 Gm. per cent on October 21, the albumin being 1.65 Gm. per cent and the globulin being 3.85 Gm. per cent.

On Oct. 21, 1950, the aspirated pleural fluid contained hydrochloric acid for the first time. The x ray revealed an increase in the fluid on the left. A diagnosis was made of a ruptured esophagus or a rupture of a portion of the stomach contained in the hiatus hernia.

On October 22, the patient was taken to surgery and an operation was carried out by Dr. A. Ray Wiley and Dr. R. M. Shepard. Through a left thoracotomy incision, a foul smelling empyema was entered and drained. The lung was decorticated and a rent in the esophagus was found 1 cm. above the diaphragm. This was closed with interrupted cotton sutures, the lung re-expanded, and the chest closed with an anterior and posterior drain in place.

The patient did satisfactorily after operation. However, one week after the thoracotomy the ruptured esophagus reopened and an esophago-pleural cutaneous fistula was formed at the posterior drainage site.

The patient was discharged from the hospital on November 31, 1950, with a posterior drainage from the esophageal fistula but otherwise in satisfactory condition. He continued to do well, with his fistula draining very little. Recent follow up is not available.

CASE 4.—J. H., a 55 year old man, was admitted to Hillcrest Hospital, Tulsa, Okla., on March 2, 1949. His chief complaint was inability to swallow. He had had this difficulty off and on for the past three months and, for the past eight weeks, had taken nothing but liquids. He had lost about twenty pounds. He had had some "heartburn" and substernal discomfort.

His past history revealed that he has had a history of trouble with swallowing for six years. In June, 1948, a biopsy of the esophagus had revealed the presence of a mild esophagitis.

On physical examination, this patient was a well developed, poorly nourished man who gave the appearance of being in constant distress. An examination of the chest revealed it to be clear to auscultation and percussion. The respiratory rate was 16. The heart was regular without thrills or murmurs, not enlarged, and his blood pressure was 110/90. The pulse rate was 68. On examination of the abdomen, the liver was found to be greatly

enlarged, extending four fingerbreadths below the costal margin. It was smooth and not tender. The spleen was not enlarged, and the abdomen showed no evidence of masses, scars, tenderness, or ascites. Thorough examination revealed no other abnormalities.

Barium swallow at this time revealed an irregular obstruction involving the lower third of the esophagus extending to within one inch of the cardia. The esophagus above was slightly dilated. The urine was normal. There were 4.2 million red blood cells and 13 Gm. hemoglobin. White blood count was 6,600 with 63 polymorphonuclear leukocytes. The blood sugar was 92.1 mg per cent, the prothrombin 91 per cent, the total protein 4.7 Gm. per cent, and the chlorides were 679 mg per cent. The albumin globulin ratio was not done.

Because the patient was unable to take sufficient food by mouth and because dilation of the esophagus at this time seemed hazardous, he was advised to have a temporary gastrostomy followed later by esophageal resection or dilatation. This he refused, so on March 3, 1949, the patient was taken to surgery. A No 8 x 45 esophagoscope was introduced down to a level just below the aortic arch. At this level it was noted that the mucosa of the esophagus was extremely red and edematous and the scope was inserted no further. Nos 10 and 12 esophageal bougies were successively introduced over a previously swallowed string. These passed under direct vision into the stomach with no difficulty. Further dilatation was not considered wise.

The patient returned to his room in good condition and later had a lunch of clear liquids. For dinner he had cream soup and other liquids. Both meals were taken without pain or difficulty in swallowing. The next morning he had a soft diet for breakfast and another soft diet for lunch. About ten minutes after his lunch, while sitting up in bed, he had a sudden excruciating substernal pain. He did not vomit.

Examination one hour later showed the patient to be writhing in pain and sweating profusely. His color was good, there was substernal and upper abdominal pain with mild abdominal rigidity. There was no subcutaneous emphysema. The pulse was 100, temperature 99.4° F and respiration 24. The blood pressure was 110/80. Breathing was difficult.

Within an hour roentgenogram of the chest revealed a collapse of the right lung and fluid within the right pleural cavity. There was no evidence of air under the diaphragm or of subcutaneous or mediastinal emphysema. A large rubber catheter was introduced through the chest wall and into the pleural cavity and suction applied. All food by mouth was stopped and a Levine tube put into the stomach through the esophagus and the patient was put on massive doses of antibiotics, oxygen, and enough morphine to relieve the pain. Under this conservative management, he did reasonably well through March 5. However, on March 6, the patient's condition was apparently deteriorating. Roentgenographic examination at this time revealed further fluid in the pleural cavity and the right lung only partly re-expanded. The drainage from the tube within the pleural cavity appeared purulent.

On March 6, 1949, the patient was taken to surgery and a right thoracotomy carried out, at which time a cleansing of the lung and pleural cavity was accomplished and a small rent at the lower end of the esophagus, approximately one and one half inches above the diaphragm on the posterior wall of the esophagus was found. The esophageal wall at this point was extremely friable. The hole was closed with some difficulty and a flap of pleura brought over the hole as an additional support. The thoracotomy incision was closed leaving two large drains in place, one anteriorly and one posteriorly. It was felt that the esophagus was in such bad condition that the perforation might possibly fail to close and for that reason a small incision was made in the anterior abdominal wall and a Witzel gastrostomy was carried out.

The patient did reasonably well following this procedure. The lung re-expanded and the patient tolerated the gastrostomy well and gained weight. The red blood count remained normal. The white blood count fell and the total protein rose to 5.5 Gm per cent. However, on the seventh postoperative day there was an increased drainage

through the posterior drainage tube which had been left in place. Roentgenographic examination at that time revealed that there was a posterior pocket leading from the esophagus out through the pleura and the posterior drainage tube.

The patient continued to do well. However, on March 18, the gastrostomy tube was inadvertently removed and it was impossible to reinsert it. Further need for it was apparent so the patient was taken to surgery and the Witzel gastrostomy re-established and the abdomen closed in the usual manner. He continued to do well and gained weight. He was discharged on March 26, 1949, with a gastrostomy tube in place and a small esophago pleural cutaneous fistula posteriorly. The patient was then transferred to a veterans hospital, where he continued to do well.

At the present time he still has his esophageal fistula and is not able to carry on his regular vocation. However, he is up and around and in reasonably good health.

COMMENT AND DISCUSSION

The four described cases have the typical history and physical findings associated with a spontaneous rupture of the esophagus. They are not spontaneous, however, because of the fact that the rupture is associated with disease of, or a change in, the esophageal wall. In two instances the disease of the esophagus was symptomatic. In the other two cases, there was history of peptic ulcer which is often associated with esophagitis. In three instances there is definite history of an inadequate diet and alcoholism. In all four cases there was a disturbance of protein metabolism with a low total protein and a reversal of the albumin-globulin ratio. All of the above factors contributed to a weakness of the esophageal wall.

Only one case was associated with vomiting but the other three were preceded by a large meal or an alcoholic bout. These conditions possibly contributed to increased intraluminal pressure in the esophagus. Two factors are responsible for the rupture in all cases: first, increase in intraluminal pressure, second, further weakening of the esophageal wall.

In none of these cases was definitive surgery undertaken at the proper time. The delay in carrying out a thoracotomy and repair undoubtedly contributed to the postoperative complications and prolonged convalescence in these cases. Fortunately they all survived in spite of delayed treatment. In one instance it is quite likely that the patient would have survived under conservative surgical management but would have been left with an empyema and an esophageal fistula. Thoracotomy and repair in this case restored the lung to its normal physiology and made possible repair of the fistula.

These cases bear out the point that if the patient survives for forty-eight hours, one may be more conservative in his methods of treatment and the patient has a reasonable chance of survival. If the patient is seen in the first few hours one cannot be at all certain that he will survive without surgical repair. They also bear out that thoracotomy and closure of the defect will bring about a more nearly complete and more prompt recovery.

A study of these cases correlated with the cases reviewed in the literature leaves no doubt that thoracotomy with repair is, in every case, the most satisfactory method of treatment.

ETIOLOGY

Rupture of the esophagus following instrumentation or as a result of a foreign body is usually diagnosed without delay^{28 33 36} The etiology is obvious and the problems are the same as in rupture of the esophagus from a less obvious cause

Rupture secondary to neoplasm or long-standing disease offers a different problem The remarks in the paper will be largely confined to "so-called" spontaneous rupture of the esophagus

"Spontaneous rupture" means rupture of the esophagus that is presumably normal before the perforation occurs and not caused by an external force It is not associated with disease in or around the esophagus, with a foreign body in the esophagus or with manipulation of the esophagus

The etiology is undecided and many ideas concerning it have been put forth^{1 13 16, 19 21 26 30 44 47} McKenzie, working on human cadavers in 1848, noted that it required $5\frac{3}{4}$ to 11 pounds pressure per square inch to rupture the esophagus of an adult, the average being approximately seven pounds per square inch Other investigators have similar results²⁴ It was also pointed out that the rupture nearly always occurs in the terminal three or four inches of the esophagus which experimentally represents the weakest portion of the esophagus^{24 29} It is the rapidity of the rise in intraluminal pressure rather than the total degree of pressure that is the most important factor in the cause of perforation²⁴

It is interesting to note that these observers found that the esophagus of a child would resist more pressure than that of an adult It requires approximately four times as much pressure to rupture the esophagus in a child below twelve years as it does to rupture an adult esophagus

From an anatomical standpoint, certain other observations by Mosher³⁰ are significant These points are (1) Esophageal muscles at the lower end terminate in a conical fashion, the tapered end being extremely thin, (2) an inherent weakness exists at the site of entrance of the vessels and nerves into the musculature at the lower end of the esophagus, (3) the esophageal muscles at the lower end are relatively weaker than the muscles of the stomach, and (4) segmental defects are found in the circular muscle layer at the lower end of the esophagus in some of the cadavers dissected

Beattie¹ noted experimentally that electric stimulus of the tuber cinereum in animals caused a hyperperistalsis in the stomach together with hyperemia of its mucosa This may be significant in accounting for the severe vomiting and occasional rupture of the esophagus that is found in patients with intracranial disease^{12 3 49}

Kinsella⁴ reviewed six circumstances in which rupture may occur All of these imply either a change in the esophageal wall or the application of a force A review of many cases in the literature brings out the facts that not a few of the cases have a history suggestive of peptic ulcer, many are chronic alcoholics, and some are in a poor state of nutrition from various causes These conditions imply changes in the esophageal wall The fact that, with one exception,

the rupture occurred in that part of the esophagus that experimentally or anatomically proved to be the weakest would lead one to believe that force plays an important part. One should not lose sight of the fact that disease is also more common in this lower portion.

Since circumstances imply that both force and previous changes in the wall of the esophagus have a major role in the cause of this condition, as suggested by Samson,⁴¹ the word "spontaneous" is a misnomer. Since the term "rupture" means perforation by force it is ill used with the word "spontaneous."

The term "postemetic" may accurately be used to describe many of these cases. However, three out of our four cases did not vomit, and several cases in the literature failed to vomit. Vomiting only *after* the severe pain following the perforation is reported several times. This suggests that the vomiting may be the result of the rupture in some cases, and not the cause. Vomiting causes rupture of the esophagus by a sudden increase in the intraluminal pressure. It has been suggested that this increase in pressure usually occurs when the stomach is full and therefore the pressure cannot be released into the larger lumen of the stomach.

In the cases reported in this paper it was noticed in each case the total proteins were low and there was a reversal of the albumin-globulin ratio. The significance of this is not known.

It is suggested, in summary, that rupture of the esophagus is caused by a sudden increase in intraluminal pressure. Nearly all of the reported cases show positive or suggestive evidence of pre existing disease in the esophagus. Such disease may have been asymptomatic before the perforation occurred. Poor nutrition or disturbance in protein metabolism may weaken the esophageal wall.

PATHOLOGY

Rupture of the esophagus is most frequently seen on the posterolateral wall of the left side in the lower eight centimeters of the organ (10 per cent on the right side²⁴). The tear is usually vertical and may be from 2 mm to 8 cm long. If the tear involves the stomach wall, severe bleeding may occur. Perforation of the middle third of the esophagus has been described only once (by Fitz) in a paper by Privitera and Gay.⁴⁰ Complete disruption of the esophagus was described in the original case by Boerhaave¹⁵ and again by Higginson and Clagett.²²

In true spontaneous rupture of the esophagus no disease process, either gross or microscopic, is demonstrable at the site of the tear. Changes that are seen are often thought to be the result instead of the cause of the rupture. It seems more often than not that they are the cause of the perforation.

Tears of the esophagus have occurred in patients with poliomyelitis,²¹ meningitis,²⁰ paresis,¹⁹ intracranial hemorrhage,⁴⁰ history of weight lifting,⁴⁰ roentgen examination of the gastrointestinal tract,²³ defecation,⁴⁰ administration of anesthesia,⁴⁰ pregnancy,⁴⁵ and brain tumor.¹³

HISTORY OF THE DISEASE

This condition is usually seen in men (86 per cent)¹⁵ between the ages of thirty-five and fifty-five years. The youngest reported case was two years,⁴

and the oldest was seventy-five years old^{10 23} These patients may be well nourished and may have enjoyed relatively good health previously A majority of the cases, if closely questioned, will reveal suggestive or positive evidence of gastrointestinal upsets and a history suggestive of peptic ulcer Often these cases are chronic alcoholics and for that reason or some other reason have not eaten a well-balanced diet

The onset of the disease is marked by a sudden, severe pain This may be preceded by vomiting, it may be followed by vomiting, or, in some instances, is not associated with vomiting The pain may be substernal, epigastric, or abdominal and, characteristically, is so severe that large doses of narcotics are required to relieve it

Within a few minutes to a few hours, the epigastric or abdominal pain will become substernal It occasionally will radiate around into the back and between the scapulae Pleural pain may occur on either side The attack is often preceded by a large meal or by an alcoholic bout The patient may point out rigidity of the abdomen, particularly the upper abdomen There is often definitely a discrepancy between the abdominal rigidity and the abdominal pain

These patients may be in shock, and complain of difficulty in breathing The degree of cyanosis and dyspnea depends on the severity of the pain and the presence or absence of fluid in the chest, of tension pneumothorax, and of mediastinal and subcutaneous emphysema The state of shock depends upon the degree of pain, the presence of hypoxia and the amount of blood loss Extreme thirst is a common symptom.

PHYSICAL EXAMINATION OF THE PATIENT

When the patient is first seen he appears severely ill and is often lying on his side with his knees drawn up He is dyspneic and may be cyanotic Shock may be present The pulse is often not greatly elevated, usually between 90 and 100 The temperature of the first few hours runs between 99 and 100 degrees the respiratory rate is always rapid, being between 30 and 40, and the blood pressure is usually normal, but may be quite low

Examination of the skin and subcutaneous tissue often reveals evidence of subcutaneous emphysema This may be generalized but is often first noted in the left supraclavicular area, the right supraclavicular area or at the supra-sternal notch It is often noted over face and neck and only rarely over the anterior chest wall

Examination of the chest may be entirely negative However, if pleural effusion is present the typical findings of lack of breath sounds and dullness over the area of the effusion can easily be noted This usually occurs on the left, but may occur on the right side or bilaterally In 71 cases reviewed by Pruneri and Gayle 91 per cent had a hydrothorax or hydropneumothorax, and 10 per cent showed subcutaneous or mediastinal emphysema or both (Table I)

The abdomen always shows rigidity, particularly in the upper abdomen, and the disease is often thought to be in the abdominal cavity This has led to the

misdiagnosis of a ruptured intra-abdominal viscus with a resulting laparotomy. Peristalsis is always reduced. The examination of the abdomen otherwise is negative.

The remainder of the examination is usually of no particular significance. One may note changes in the reaction of the pupils if the patient has had narcotics for the relief of pain. The reflexes are hyperactive throughout.

LABORATORY EXAMINATION

Examination of the blood will nearly always show an elevation of the red cell count due to dehydration. Occasionally anemia is noted because of hemorrhage secondary to the rupture. Ordinarily such a change in the blood count has not had time to occur even though bleeding may be severe. The white blood count shows a slight relative increase in polymorphonuclear leukocytes with leukocytosis.

A roentgenographic examination of the chest taken in the lateral decubitus and/or upright position will reveal no air under the diaphragm. This is an extremely important finding because no case of rupture of the esophagus has shown air beneath the diaphragm.⁴⁰ The chest roentgenogram will often show fluid and free air within the pleural cavity. Subcutaneous emphysema may be a prominent finding in the chest roentgenogram. A fluid level or emphysema within the mediastinum is pathognomonic (Table I).

TABLE I FINDINGS IN EIGHTY SEVEN CASES OF RUPTURE OF THE ESOPHAGUS

Hydrothorax	85%
Emphysema	62%
Hydrothorax (Left only)	60%
Hydrothorax (Rt only)	33%
Hydrothorax (Bilateral)	7%
Air Beneath Diaphragm	0%

DIAGNOSIS

The diagnosis of this condition can often be made with ease from the history and physical examination. A diagnostic triad has been emphasized by Barrett and others.^{3 4 9 12 15} This triad is (1) Rapid respiration, (2) abdominal rigidity, and (3) subcutaneous emphysema. Kinsella²⁴ states that the diagnosis can sometimes be made over the telephone.

If the diagnosis is in doubt, other diagnostic proceedings may be carried out. These consist of aspiration of the fluid within the chest cavity. This fluid may be turbid or blood stained. It should be tested for food particles and hydrochloric acid. If either is present, the diagnosis is further substantiated. Often the odor of the fluid is important. A swallow of barium or lipiodol followed by fluoroscopic and roentgenographic examinations will often outline the perforation or show a periesophageal pocket with direct communication into the pleural cavity.

If one is still in doubt, dye can be taken by mouth and recovered in the intrathoracic fluid, thus establishing the presence of a fistula between the esophagus and pleural cavity.

DIFFERENTIAL DIAGNOSIS

Ruptured esophagus is most frequently confused with ruptured peptic ulcer, acute pancreatitis, or coronary thrombosis. In addition to these, this condition may simulate mesentery thrombosis, dissecting aortic aneurysm, pulmonary embolism, spontaneous pneumothorax, rupture of a bronchus, volvulus, intestinal obstruction, strangulated bowel, rupture of a gall bladder, and diaphragmatic hernia associated with rupture of the stomach or bowel. If the patient relates the usual history, exhibits the diagnostic triad mentioned above, has fluid in the chest, and lacks free air under the diaphragm, most of the other confusing conditions can be quickly ruled out. Most diagnostic failures are due to the fact that the examiner is unaware of the symptoms of rupture of the esophagus or simply fails to think of the possibility.

Mesenteric thrombosis, coronary thrombosis, perforated intra-abdominal viscus, intestinal obstruction, strangulated bowel, and volvulus, seldom, if ever, cause fluid in the chest and are never the cause of subcutaneous or mediastinal emphysema, and, therefore, should be easily ruled out if one or both of these conditions are present. Some of these conditions are frequently associated with free air beneath the diaphragm, a finding always absent in esophageal rupture.

Rupture of the gall bladder may cause fluid in the chest and is rarely associated with air beneath the diaphragm, however, it is frequently associated with a previous history of gall bladder disease. There is often an obvious gall stone seen in the roentgenogram and it is never associated with subcutaneous emphysema. Acute pancreatitis can usually be ruled out by determining the level of the blood amylase. Acute pancreatitis never causes mediastinal or subcutaneous emphysema but may cause pleural effusion. It is often associated with severe abdominal pain, rigidity and shock.

Pulmonary infarction or embolus never causes emphysema, but they are often associated with a cough and hemoptysis which are not seen in rupture of the esophagus. Abdominal pain and rigidity are not present. The pain and shock may be severe.

Spontaneous pneumothorax may produce any of these symptoms found in rupture of the esophagus but as a rule does not cause the severe pain or prostration associated with rupture of the esophagus. There is usually a high degree of collapse of the involved lung, and fluid rarely occurs at the onset but makes its appearance in twenty-four to forty-eight hours. Subcutaneous emphysema is rarely seen in this condition.

Rupture of the bronchus may cause severe subcutaneous and mediastinal emphysema with pain. There may be a collapse of the lung with severe dyspnea, shock, and prostration. Fluid formation does not occur as an early finding but may form later.

Neither of the last two conditions mentioned are commonly associated with the symptoms of vomiting, retching, or other gastrointestinal symptoms. They rarely cause epigastric pain and they are never associated with a board-like rigidity of the abdomen.

Rupture of a previously herniated stomach or bowel into the mediastinum with an extension into the pleural cavity may simulate rupture of the esophagus in every respect. Careful fluoroscopic and roentgenographic examinations, during which the patient swallows some barium or lipiodol, will often demonstrate the herniated stomach and suggest the diagnosis. Since the treatment of this condition is the same as the treatment for ruptured esophagus, the differentiation of these two conditions is only of didactic importance. In either case, one is forced to do an immediate operation with closure of the rupture.

PROGNOSIS

The prognosis of rupture of the esophagus is always extremely grave. In a review of fifty-three cases by Kinsella,⁴ thirteen died in less than twelve hours, twenty-four in less than twenty-four hours, eight in less than forty-eight hours, and only eight survived over forty-eight hours. Early deaths are due to shock, tension pneumothorax or, possibly, hemorrhage. Later deaths are due to pleural or mediastinal infection. In 1946, Eliason and Welty¹³ reported a mortality of 100 per cent in these cases with the exception of the two cases reported by Graham.²¹ Thirty of the 108 cases considered in this report recovered. All recoveries followed surgery.

While the prognosis may be influenced by the size and position of the perforation, death is likely to occur unless the diagnosis is made and adequate treatment is carried out at the earliest possible time.

In these days of plentiful antibiotics, of the judicious use of blood and fluids, and of prompt surgical intervention, the patient can be offered a far better prognosis.

TREATMENT

Treatment of rupture of the esophagus may be divided into conservative management^{12 16 37 45} and radical management,^{3 4 10 15 24 27 41} or better, by surgical drainage as compared to thoracotomy and repair. Samson,⁴¹ in his recent article, compares fifteen cases treated by the two methods. Of the fifteen cases treated by thoracotomy, ten survived with lesser morbidity. Only eight of the fifteen treated by drainage survived and these with a higher morbidity. The latter group had more postoperative complications and much longer hospital stay. Of the twelve cases reported in 1951, nine underwent thoracotomy and eight survived. Three who were not operated upon, died. The four cases reported in this paper all underwent thoracotomy and survived.

This condition is one of the definite surgical emergencies. If the diagnosis is not obvious, steps should be taken to establish it immediately. Only those procedures that are absolutely imperative to make a diagnosis should be done. The patient should be operated upon as soon as the diagnosis is established. A thoracotomy and repair of the rupture should be carried out. Nothing is to be gained by procrastination.

The presence of shock should not delay surgery. The shock is treated and the thoracotomy undertaken. With proper anesthesia and controlled

respiration the chest is opened. The pleural cavity should be aspirated and the lung re expanded. The rent in the esophagus is to be found and closed with two layers of nonabsorbable suture. A feeding tube should be passed down the esophagus beyond the perforation and into the stomach. This may also be used for decompression. The chest should then be closed leaving in drainage tubes at appropriate places to insure complete expansion of the lung, adequate drainage of the pleural space, and direct drainage from the site of repair in case of a breakdown.

The shock in these cases is due to pain, hypoxia, and blood loss. The hypoxia is due to tension pneumothorax, pleural fluid, and poor respiration as a result of pain. Under adequate anesthesia the patient receives high oxygen intake, with the pain stimulus gone, respiration improves. As the chest is entered the tension pneumothorax is relieved, the pleural fluid is removed, and the lung is expanded. Before and during surgery, the blood loss is replaced. As the causes of shock are relieved the patient improves and is seen to tolerate the procedure well.

Postoperative complications are empyema, mediastinitis, and breakdown of the repair. The mediastinum is left open with opportunity for adequate drainage into the pleural cavity and, thus, to the outside. Thorough cleansing of the area around the perforation and of the pleural cavity and use of antibiotics in the pleural cavity as well as systemically are imperative. Prompt and complete expansion of the lung is important in preventing empyema. The repair may break down from the sixth to the tenth postoperative day. If this occurs, drainage of the open perforation is necessary. The fistula thus formed will often close in several weeks.

Following surgery the esophagus is kept at rest for a week or more and the patient is fed through a Levine tube or parenterally. Gastrostomy is seldom indicated. Because of changes in the tissues at the site of perforation, healing is often retarded and thus requires a longer period before feeding by mouth is safe.

If other complications make thoracotomy intolerable for the patient and a more conservative method of management indicated it may be undertaken. Shock should be combated with blood and fluids. Drainage should be established to relieve tension pneumothorax and to withdraw pleural fluid. Negative pressure should be maintained to assure complete and rapid expansion of the lung. Narcotics, sedatives and intercostal block are used for relief of pain. Antibiotics are used in high dosage. A Levine tube or gastrostomy may be employed for feeding and decompression purposes. The esophagus is put at complete rest. If mediastinal emphysema is causing respiratory distress this can be relieved by making an incision under local anesthesia just above the suprasternal notch through the anterior fascia and muscles of the neck. Through this incision a catheter can be introduced into the mediastinum for decompression.¹¹

Should the patient survive for forty eight hours or more without surgery, the conservative methods of treatment may be satisfactory. In any event,

more conservative methods are often associated with residual fistulae and empyema. These require thoracotomy and correction later.

SUMMARY

1 The etiology, pathology, symptomatology, diagnosis, and treatment of rupture of the esophagus are discussed

2 It is suggested that the term "spontaneous" is a misnomer in most cases.

3 The findings are typical in most cases and the diagnosis not too difficult. A diagnostic triad is emphasized.

4 Most diagnostic failures are due to lack of knowledge or failure to think of the possibility of rupture of the esophagus.

5 It is felt that the treatment of choice is immediate thoracotomy with closure of the perforation and re-establishment of the normal anatomy and physiology.

6 With early diagnosis and treatment the prognosis is good.

7 Four cases are reported. All were treated successfully.

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DISCUSSION

DR. OSLER ABBOTT, Atlanta, Ga.—I certainly feel that Dr Anderson is to be congratulated. I also feel very strongly that we owe a tremendous debt of gratitude to our secretary for organizing our thinking in regard to this disease. I hesitate to rise, but I feel in some ways it is my duty when I can add to the series 31 patients with spontaneous rupture of the esophagus. I would like to show slides that exemplify some of the lessons we have learned in this regard.

(Slide) I think it is important to stress what appears to be practical classification of patients, and there are some things in the symptomatology that are important. These patients are not all in the lower income brackets. The ones in the upper income brackets are oftentimes ulcer patients and have a restricted diet. We had the opportunity, I should say not too much desired, to take care of the brother in law of a famous stellar surgeon, and cure him of this disease. The first thing I would like to emphasize in regard to the symptomatology, is that you must listen to the patient, because in a review of the history of patients who died or had delayed treatment, they so often give the bizarre history that something ruptured and moved into the chest, or something ruptured in the chest and moved. This is a very important differential diagnostic symptom.

(Slide) This is from the paper by Drs Fincher and Swanson on the neurogenic causes of this disease, which appeared in the *Annals of Surgery*. Of course when you study anything neurosurgical, you look back in the textbooks, and usually find that Dr Cushing described it in detail thirty years ago. The important thing is that these patients, after mid or posterior fossa surgery, and possibly a majority of patients who die following neurosurgery, may have this as a complication.

The management of such patients I think requires three things. First, consciousness of the disease, second, any patient following a neurosurgical operation, who has blood in the initial vomitus post-surgery, has this disease, and next, the patient must have ventricular drainage to control the pressure on the tuber cinereum area, and, finally, if they are operated upon transpleurally, there is diffuse disease of the esophagus, and Dr Torek's T tube in the esophagus is the answer to this type of patient. I think we should compliment him on that.

(Slide) This is a typical example of rupture in a neurogenic case and one of the factors of course is weakness of the lower esophageal musculature and the attachment at the diaphragm and periesophageal fibrosis. I think that is important in the mechanism.

(Slide) One of the things I would like to add to conservative management. In patients who are seen more than 15 hours after onset of the disease, it is valuable to put in multiple thoracotomy tubes and irrigate with saline until the pH of the pleural fluid is within normal limits. This will relieve the secondary shock if the mediastinal pleura is not under tension.

DR WILLIAM W HEROY, Huntington, N Y—I feel that Dr Anderson has given us a very clear picture of this interesting problem. I wonder whether these ruptures are really spontaneous, or, for the most part, are the result of underlying disease. It is a bit unclear in my mind why we do not have, in the cases reported, some underlying ulcers. Many of the patients we see with this condition will give histories of chronic pain under the sternum. I would like to show two cases to exemplify this point.

(Slide) The first case was a 33 year old chronic alcoholic hotel maid, who was in the habit of drinking large quantities of beer. For lunch she claimed to have had several beers, and returned to work where she developed severe substernal pain. In order to relieve this pain she took a dose of soda bicarbonate and added a little vinegar to make it more potent. Following this dose she was seized with overwhelming pain in the substernal area and was rushed to another hospital, where she was kept for a couple of hours until she vomited about 200 cc of blood, and was transferred to us. When she arrived she showed the classic picture of just what she had, in its most severe form with shock and subcutaneous emphysema.

(Slide) Because of her very poor condition at the time we finally got her, it was elected to wait, and she was given supportive care and very heavy antibacterial therapy. She then went into a toxic psychosis and operative procedure was further delayed until she developed evidence of empyema in the left chest which was explored and drained one week after admission. She did fairly well until a week later, when she developed a cutaneous fistula, and the feedings given by Levine tube dumped out. A gastrostomy was done to control this, but the gastrostomy feedings came out through the fistula. Over a period of the next two weeks the fistula gradually closed, and she made an uneventful recovery. After that, and a year later she had a perfectly normal child. At the time of the gastrostomy the liver biopsy showed fatty disease of the liver.

(Slide) This simply shows the fistulous tract running out to the chest wall.

(Slide) This shows the final film.

(Slide) The second case is a 68 year old man who, while watching television one night, was seized with severe epigastric pain. There was a history of two weeks of substernal pain and it was thought that he had a perforated ulcer. He refused hospitalization at first, but finally came in and was explored. The entire abdomen was negative. The esophageal hiatus

was explored with the finger, but the peritoneum was not opened. If you do not open the peritoneal surface and have a collection in that area, it may be missed as it was in this case

(Slide) This last slide shows the collection, which was actually well localized between the diaphragm and the pleura, and I think gives the whole picture away. This is emphysema before it has gotten into the mediastinum, before it has entered the pleural space. I would go along with Dr. Anderson 100 per cent that one should look for esophageal perforation and operate early and repair the defect.

I would like to ask him if he does not feel that a great many esophageal perforations have pre-existing and unrecognized underlying ulcers.

DR. RICHARD H. OVERHOLT, Brookline, Mass.—I think we will all agree that it is important to carry out a direct surgical attack on the situation pointed out by Dr. Anderson and do it as early as possible if surgery is required. However, sometimes the surgeon has to make the best terms he can with a difficult situation and cannot control the timing of treatment. I would like to report a rather unusual case in which the etiology was related to acute alcoholism. There was a long interval between esophageal rupture and mediastinal drainage. During this interval, the same liquid that precipitated the rupture was the only nourishment taken and probably favorably influenced the outcome. This patient was on a hunting trip and, because there were no deer the first day, he began drinking. Late in the evening there was some scuffling and wrestling and finally he ended up at the bottom of a pile of hunters. Soon his friends noted marked swelling of his neck and face. Swallowing was painful. On the following day, while the rest of the men went out hunting, he stayed in bed, sipping Scotch whisky. He was there eight days and on the return trip, the patient was so critically ill that his friends took him to a hospital where he remained for four days.

(Slide) This roentgenogram shows a large air collection and fluid levels on both sides. There was diffuse density in both lung fields, indicating bronchopneumonia.

(Slide) A barium swallow was given which revealed an esophageal communication with both pleural cavities and a massive bilateral emphysema. Closed drainage was provided after bilateral rib resection and aspiration of purulent material and food contents. Due to the marked inflammatory reaction everywhere, no attempt was made to repair the esophageal defect. There was a rent about 7 centimeters in length. The patient carried and was fed through a Levine tube for 67 days.

(Slide) There is satisfactory expansion of the lung and there has been a check on esophageal function five years later. There is no stricture of any kind. This illustrates that you must not give up on some of these very serious cases, and we have to make the best terms we can with them, regardless of the time factor.

DR. JAMES M. MASON, III, Birmingham—I have been much impressed with the progress that has been made in the care of perforations of the esophagus since it has been demonstrated that they can be successfully treated by primary one-stage operation.

Sweet has demonstrated that excellent results have followed one-stage closure of esophageal diverticula, and his work has no doubt influenced the one-stage primary closure of esophageal perforations. Many of us have had excellent results with primary closure, whereas late attempts at closure have been disappointing.

DR. HERBERT WILLY MEYER, New York—I think it is most timely to have heard the excellent paper by Drs. Anderson and Curtis. I would like to report two cases. They are not my own cases as they date back a great many years. In 1940 I had the occasion to look up the literature on perforations of the gastrointestinal tract. In 1724 Blauwe reported a unique case in which the rent in the esophagus was transverse. The majority of spontaneous perforations of the esophagus are linear. It is important, however, that we keep in mind that spontaneous perforations may also be transverse in location. This emphasizes Dr. Anderson's advice that early operation is certainly indicated and indicated.

It is of historic interest also to quote a case by MacKenzie published in his book *Manual of Diseases of the Nose and Throat* (London, 1884) "The patient, Baron de Wassenauer, was 50 years of age and, with the exception that he had a sense of fullness after moderate meals, was in perfect health. To relieve this disagreeable feeling he was in the habit of taking a copious draught of an infusion of 'blessed thistle' and ipecacuanha. One day, about 1 30 in the evening, when he had taken no supper but had had rather a hearty dinner, he was bothered by a peculiar sensation in his stomach, and to relieve this he swallowed three tumblersful of his usual infusion. But to no avail. He then tried to excite vomiting by tickling the fauces. In retching he suddenly felt a violent pain. He diagnosed his own case by saying that there was 'the bursting of something near the pit of the stomach.' He became prostrate and died in 18 hours. At necropsy, it was seen that without any previously existing signs of disease the esophagus had been completely rent across in a transverse direction."

There is no question that immediate surgery is indicated in practically every case of suspected or proved spontaneous perforation of the esophagus.

DR OWEN H. WANGENSTEEN, Minneapolis, Minn.—I rise to a point of definition. The word "spontaneous" still hovers over our concept of this disease. However, I think the evidence is good now that we should capitulate to the title "acid peptic perforation" of the esophagus. Whereas there are occasional conditions such as Dr Heroy enumerated, and there are hydrostatic phenomena involved, I submit that *they are quite secondary*. The cecum and the stomach are the two large viscera with large diameters. The stress or tension on any surfaces to which intraluminal pressure is applied is the product of the pressure times the diameter times π (T [tension] = Pr [pressure] $\times D \times \pi$).

Whereas the fundic end of the stomach or the cecum can be ruptured at pressures of 80 mm. of Hg, it takes more than an atmosphere of pressure to rupture the esophagus (approximately 800 mm Hg).

If one ligates the pylorus in the dog, maintaining fluid and electrolyte balance parenterally, within four days, perforation of the esophagus was observed in 4 of 8 dogs subjected to this experiment, esophagitis was observed in all the eight dogs, the stomach, as you observed from the slides, was quite normal in all.

Moreover, when acid peptic juice collected from an isolated dog's pouch was dripped into the esophagus at a maintained intraluminal pressure of 20 cm of fluid, a severe esophagitis with perforation always followed. In fact, the cat's esophagus can be perforated within 20 minutes by dripping acid peptic juice into it under these conditions (Ferguson and associates, *Surgery* 28: 1022, 1950).

Furthermore, the esophagus also is sensitive to the other digestive enzymes of the upper alimentary tract, even the reflux of bile and pancreatic juice into the esophagus will cause erosion of the esophagus, even after total gastrectomy. In the dog, anemia is observed following this occurrence from the erosive esophagitis (Cross and associates, *Proc Soc Exper Biol & Med* 77: 862, 1951).

Finally, I want to show one of Dr Harvey Cushing's pictures illustrating an erosive action of the digestive juices upon the esophagus. It is to be remembered that Cushing emphasized the relationship of the interbrain to peptic ulcer in the Balfour Lecture, in fact, that was the title of the lecture (*Surg, Gynec & Obst* 55: 1, 1932).

I submit it is the reflux of acid peptic juice into the esophagus which was the likely cause of the perforations observed by Dr Cushing.

I think it is time that we delete the word "spontaneous" and call this disease by its rightful name—acid peptic perforation of the esophagus.

DR DAVID H. WATERMAN, Knoxville, Tenn.—I rise merely to report six cases of perforation that we have treated. In two of these the diagnosis was confirmed by aspiration of beer from the chest. Insofar as we know, television was not etiologic in any of our cases.

I certainly subscribe to the use of immediate exploration. I might say that at the time our cases occurred, however, we were a little more timid about opening the thorax than we are today. In all except the first patient, who died promptly after admission with bilateral pneumothorax and a typical picture of shock, the patients were treated by closed thoracotomy, massive antibiotic therapy, and by later gastrostomy. All five recovered.

DR J D FRYFOGLE, Detroit.—I wish to report a case of unusual spontaneous etiology, and this case was definitely not an alcoholic. I was called to see a newborn baby, a female infant of normal pregnancy and normal delivery, who breathed and cried spontaneously at birth. However, in a very few minutes she became quite dyspneic and cyanotic and efforts at tracheal aspiration and aspiration of the hypopharynx were not successful in relieving the condition. Physical and roentgenologic examination confirmed the diagnosis of tension pneumothorax, which was relieved by needle aspiration. The baby was placed in oxygen and in several hours the crisis again arose. This time more definitive measures were carried out, and a 17 gauge needle, fashioned after the manner of the "S" needle, was placed into the chest under water seal suction. This seemed to control the respiratory difficulty. The baby was given her first feeding, of plain water, about 12 hours later and another supreme crisis arose, with everything getting black and no breath at all.

I was available at the time, and with a large syringe I aspirated the tube already in the chest and withdrew mucus. I had the horrible thought that I was merely deflating the stomach that was in the right chest. I was proved wrong on both diagnoses, however. We gave the baby a swallow of lipiodol, which transversed a normal length of the normal lumen of the esophagus right down to the diaphragm and then deviated markedly to the right and up into the right chest, so we proceeded with thoracotomy and, opening the baby's chest, we found a total mucoid empyema which occurred at about 12 hours, and a rupture at the base of the esophagus, perforating through the right pleura. I worked through the little hole, to find that there was a web directly across the base of the esophagus which did not even permit air to enter the stomach. Had I been more astute I probably would have recognized that there were no air bubbles in the stomach on the first film. The rent in the esophagus was enlarged, the small web cut out, a tube passed through the nose into the stomach, the perforation was closed, the lung decorticated and the patient did very well.

DR GABRIEL P SFLEY, New York—I would like to report an experience with conservative management of a case of ruptured esophagus. This patient was a 64 year old obese housewife, who presented herself to a gastroenterologist for vague lower abdominal pain. As part of the work up he attempted to pass an Ewald stomach tube. As he attempted this the patient violently resisted and retched. However, he persisted in his efforts and shortly afterward the patient complained of pain in the lower back. This pain was very severe and of a burning nature. Within the next few hours she experienced increasing pain, especially on swallowing, even swallowing her own saliva. Within six hours she was febrile and the following day was hospitalized and treated by oral sulfonamides and fluids by mouth. The temperature gradually rose until it reached 104° F and her general condition was serious.

I first saw her on the fifth day. At that time she was quite ill, and the roentgenogram showed a diffuse mediastinal and subcutaneous emphysema and bilateral pleural effusion. Eleven days had elapsed we elected to treat this patient conservatively. A bilateral pleural tap was done. Clear fluid was obtained from the right side, and cloudy fluid from the left. Penicillin solution was instilled. Smears of the fluid from the left side showed no types of bacteria. There were no bacteria in the right pleural cavity. Penicillin, streptomycin and aureomycin were given parenterally and nothing was allowed by mouth. She was fed intravenously. She had a perfectly uneventful recovery and at the end of one week a roentgenogram with dye contrast medium showed a perfectly normal esophagus. I mention this case because it illustrates a different approach to the treatment

DR ANDERSON (Closing) —I certainly want to thank all the discussors

With regard to the question about ulcer, in the cases considered in this paper it was difficult, because of the inadequate information concerning the cases, to really know about ulceration. I can say that a few cases had proved ulcers of the gastrointestinal tract, that is, peptic ulcers. In only one case was I able to find mentioned an esophageal ulcer that had perforated. Suggestive evidence of peptic ulcer is common in these cases.

I think it is interesting to hear about the baby with perforation. This perforation was associated with a definite congenital abnormality. Perforations of the esophagus in children are rare for two obvious reasons. (1) they do not have peptic esophagitis, have not had time to develop that, and (2) it takes four times as much pressure to rupture the esophagus of a child under 12 years of age as it does the esophagus of an adult, which may be a factor to substantiate its rare occurrence in children.

So far as treatment of late complications is concerned, I believe there is a time when conservative management is sometimes the management of choice, if the individual is seen several days after the onset and appears to be holding his own. In the case Dr Overholt mentioned, it appears to me that the man probably could not be killed by anything, after all that. I do not mean that as a reflection on Dr Overholt's treatment. Nevertheless, I think in some cases one must treat them in a more conservative manner. This phase of treatment will be discussed further in the published manuscript.

THE EFFECTS OF ATELECTASIS ON PULMONARY BLOOD FLOW IN THE DOG

RICHARD M. PETERS, M.D. (BY INVITATION), AND

ALBERT ROOS, M.D. (BY INVITATION)

ST. LOUIS, MO

IT HAS been known for some time that atelectasis of one lung results in diminished blood flow to that lung. In dogs anesthetized with barbital Moore¹ introduced two cannulae into the trachea and left main-stem bronchus by means of a left thoracotomy. After closure of the chest and removal of air from the pleural space one of the tubes was obstructed while the dogs spontaneously breathed oxygen through the other lumen. Fifteen to seventy-five minutes later, the ratios of flow through the two lungs were computed from mixed venous and arterial blood, assuming full saturation of blood draining the normal lung. Flow through the atelectatic lung amounted to 0 to 42 per cent of total flow. Similar data were obtained by Andrus². The present paper is concerned with a reinvestigation of this problem studying changes in flow as a function of duration of atelectasis under both acute and chronic conditions. In addition changes in pulmonary vascular resistance resulting from acute atelectasis were investigated.

METHODS

1. Acute Experiments—A left thoracotomy was done under Nembutal or chloralose morphine anesthesia on dogs after an intratracheal cannula had been tied in place. Artificial respiration was given by means of a Stirling pump. A large tie was placed around the left main-stem bronchus. The pericardium was opened to expose the pulmonary artery. The posterior inferior mediastinal pleura was also incised to expose the right inferior pulmonary vein. The systemic arterial pressure was constantly recorded with a mercury manometer. During a control period of ten to twenty minutes of bilateral oxygen breathing, the oxygen consumption was determined by the conventional spirometric technique. Immediately thereafter, pulmonary artery pressure was recorded by means of a Sanborn capacitance manometer attached to a needle in the pulmonary artery. Pulmonary arterial and systemic arterial blood samples were drawn for analysis of oxygen content. The left main bronchus was then occluded by tying the ligature. With the nitrogen washed out by previous oxygen breathing, the lung shrank visibly and was completely atelectatic in three to five minutes. At periods varying from ten minutes to two and one-half hours, determinations of the oxygen consumption and of the pulmonary artery pres-

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sure and oxygen content of pulmonary arterial and femoral arterial blood were repeated. In addition, the oxygen content of blood from the right inferior pulmonary vein was measured. It was assumed that this was representative of all of the blood draining the right lung. Oxygen analyses were performed on the Van Slyke-Neil manometric apparatus—duplicate determinations checked with in 0.2 volume per cent.³

From these data cardiac output and blood flow and vascular resistance of each lung during the period of atelectasis were calculated. The computation is illustrated in Fig 1, in which the pulmonary circulation is diagrammatically represented. The total flow was determined by application of the Fick principle (first and last equations, Fig 1). It was assumed that

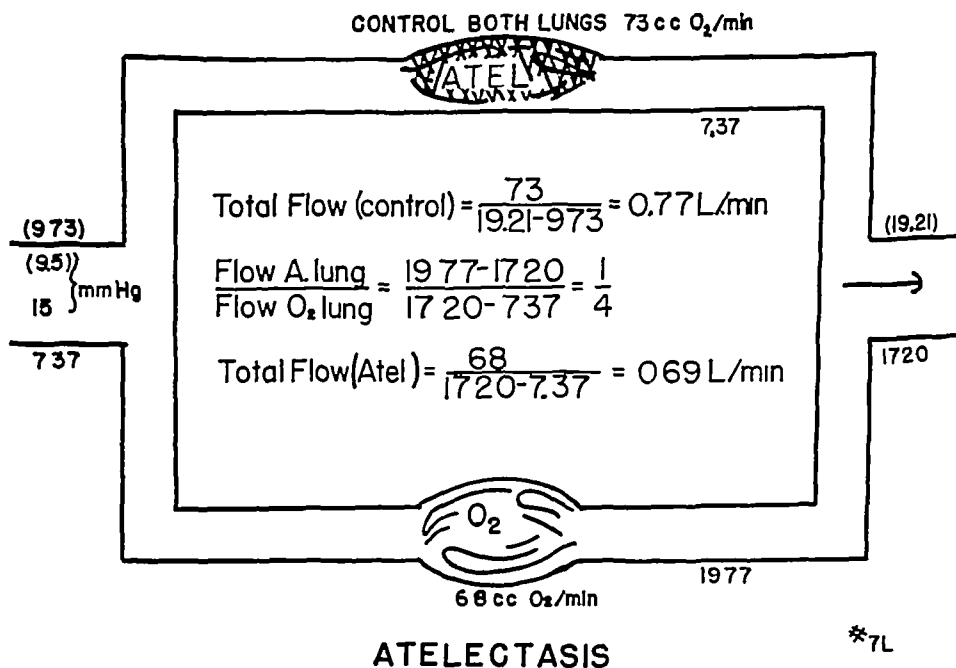


Fig 1—Diagram of pulmonary circulation showing pulmonary artery and its two branches, lungs, two representative pulmonary veins and a systemic artery. Figures between parentheses were obtained during bilateral oxygen breathing; those without parentheses during atelectasis. The unspecified figures refer to oxygen content (volumes per cent). For calculations see text.

mixed venous blood passed unchanged through the atelectatic lung. In the nine studies in which pulmonary venous blood from the atelectatic lung was collected as well, this assumption was confirmed. The ratio of flows through the lungs during atelectasis can be calculated from the oxygen content of mixed venous blood, pulmonary venous blood from the oxygen breathing lung, and mixed arterial blood (second equation, Fig 1). The pulmonary vascular resistance of each lung expressed in arbitrary units was derived by dividing mean pulmonary arterial pressure in millimeters of mercury by the value for flow

TABLE 1. VENTILATION

PULMONARY BLOOD FLOW	TIME (MIN.)	OXYGEN CONTENT—VOL. %			OXYGEN CAPACITY (VOL. %)	SATURATION (PERCENT)	OXYGEN CONSUMPTION (O.C./MIN.)	PULMONARY BLOOD FLOW (L./MIN.)	CARDIAC OUTPUT (L./MIN.)	PULMONARY ARTERIAL PRESSURE (MM. Hg)		
		ARTERIAL	VENOUS	MIXED						S	D	M
0	0	11.51	9.12	0.02	13.63	>100	70.5	34	1.44	20	0.2	13
left	10	11.11	9.73	0.02	11.88	>100	74.8	34	1.46	30	13	18
7	0	19.21	10.77	0.73	18.41	>100	73	20	77	14	8	9
left	50	17.20	7.37	0.02	10.11	>100	68	20	69	20	10	15
5	0	16.61	9.02	0.02	15.88	>100	117	8	1.75	23	11	17
left	15	16.00	9.02	0.02	10.58	>100	67	8	96	24.1	11	17
0	0	17.28	9.10	0.10	15.07	>100	103	20	1.26	23	10	15
left	05	21.31	15.08	0.10	21.80	>100	83	20	1.08	20	10	17
0	0	22.03	16.16	0.10	22.80	>100	76	20	1.50	20.2	11	17
left	05	22.78	16.61	0.10	22.50	>100	70	20	1.04	22	11	15
10	0	20.36	13.94	0.10	18.65	>100	148	0	2.3	29	10	17
left	10	20.52	13.16	0.10	18.85	>100	140	0	1.9	32	13	20
12	0	20.11	13.46	0.10	20.35	>100	170	22	2.6	34	14	22
left	30	16.89	10.73	0.10	17.90	>100	72	31	1.46	32	11	20
120	0	17.14	10.17	0.10	18.58	>100	50	29	80	41	21	26
11	0	20.21	10.11	0.10	18.30	>100	45.6	29	1.78	30	13	25
left	15	18.29	11.79	0.10	19.75	>100	73.5	5	1.31	27	13	19
11	0	19.12	15.27	0.10	17.30	>100	50	12	2.86	32	13	22
left	10	15.15	15.95	0.10	18.10	>100	114	26	5.3	22	12	15
15	0	18.01	13.28	0.10	18.00	>100	123	21	2.6	24	12	14
left	15	17.81	13.08	0.10	16.90	>100	123	12	1.44	23	10	20
10	0	20.01	12.36	0.10	20.10	>100	123	12	1.44	23	10	20
left	32	18.11	12.36	0.10	17.95	>100	123	12	1.44	23	10	20
18	0	22.56	11.90	0.10	21.50	>100	123	12	1.44	23	10	20
left	45	17.73	11.98	0.10	17.80	>100	123	12	1.44	23	10	20

* Control determination
 † 100% re-expanded between two determinations
 ‡ Nitrogen breathing, before atelectasis

TABLE II CHRONIC ATELECTASIS

DOG NO SIDE	TIME POST OPERATIVE (DAYS)	OXYGEN CONTENT (VOL PER CENT)		OXYGEN CAPACITY (VOL. PER CENT)	SYSTEMIC ARTERIAL O ₂ SAT (PER CENT)	FLOW EXPER. LUNG (PER CENT TOTAL)
		SYSTEMIC ARTERIAL	MIXED VENOUS			
85A	C*	12.52	9.12	13.16	93.0	
left	2	11.55	9.09	13.57	85.0	35
	18	7.84	4.81	10.06	77.0	33
6	C	12.30	7.95	13.65	89.0	
left	2	11.77	7.09	14.24	82.0	19
85	C	17.66	13.83	17.97	97.1	
left	3	13.15	6.42	13.78	93.4	8
18	C	13.70	10.00	14.14	95.6	
left	3	16.07	8.81	17.11	92.9	8
	10	15.06	8.50	16.48	90.4	12
	13	10.16	4.87	12.75	81.0	23†
	32	13.54	6.25	14.09	94.9	2‡
	198	12.65	8.84	14.56	89.4	16
C124	C	11.26	6.53	11.97	92.7	
left	2	9.18	4.53	11.40	79.4	26
147	C	17.66	12.32	18.17	96.0	
left	3	13.88	8.43	18.26	75.2	41
	10	12.75	8.01	14.77	85.2	29
	132	15.37	11.70	17.33	87.8	25
	165	12.06	7.11	13.90	86.6	20
12	C	13.76	9.13	16.68	91.1	
left	2	11.09	5.70	13.05	84.2	19
10	C	15.11	11.72	15.69	96.2	
left	1	13.61	7.84	15.03	89.8	12
	5	9.92	5.12	11.31	86.4	17
	8	9.80	3.80	10.58	97.8	0
90	C	11.43	9.08	11.85	96.0	
right	3	12.97	7.09	15.75	81.7	26
76	C	14.51	10.55	15.03	95.3	
right	3	10.03	5.91	12.52	83.8	24
	6	13.23	8.21	14.34	91.2	20
	14	14.26	8.22	15.74	89.7	17
13	C	16.79	11.59	17.40	96.0	
right	5	11.30	6.44	13.92	81.0	32
	12	9.99	7.21	12.01	82.0	39
	33	16.61	11.11	19.91	82.8	38
	41	15.75	8.90	19.46	80.3	32
	51	14.37	8.10	18.36	77.6	35
	55	13.86	9.93	15.36	89.5	22
5	C	13.62	8.18	14.37	93.0	
right	2	11.87	7.30	14.36	82.0	27
	7	11.47	9.99	13.75	82.5	38
15	C	12.12	9.38	12.52	95.4	
right	2	13.29	7.20	14.18	92.5	7
	8	12.90	7.42	13.06	98.0	0

*Control

†Dog pregnant

‡Partial pneumothorax on normal side

through each lung in liters per minute. This assumes that left auricular pressure remains constant and that the pressure drop across the lung is proportional to blood flow.

2 *Chronic Experiments*—Under Nembutal anesthesia, control right heart and femoral arterial blood samples were drawn for determination of oxygen content and saturation. A right or left thoracotomy was then performed and the main-stem bronchus transected and sutured. At periods varying from two days to six months the blood sampling was repeated. It was assumed that the

TABLE III. CHANGES IN PULMONARY RESISTANCE DURING ATELECTASIS

EXPERIMENTAL No.	RESISTANCE IN ARBITRARY UNITS*		FACTOR OF INCREASE IN RESISTANCE		MINUTES
	CONTROL (O.) LUNG	ATELECTATIC LUNG	CONTROL (O.) LUNG	ATELECTATIC LUNG	
6L	16	20			Control
	20	40	1.25	2.00	40
7L	23	28			Control
	27	108	1.17	4.20	50
8L	17	21			Control
	18	212	1.05	10.0	45
9L	29	35			Control
	14	56	0.45	1.6	65
10L	13	16			Control
	10	Compl Obstr	0.77		10
	11	40	0.84	2.5	60
13L	19	24			Control
	22	66	1.05	2.81	45
14L	9.6	11.6			Control
	3.5	10	0.36	0.86	40
	9.7	32	1.01	2.73	50

*Resistance = mean pressure drop across lung (mm. Hg)/flow through lung (L./min.)

blood from the atelectatic lung was the same as mixed venous blood, while that from the normal lung had the same saturation as the arterial blood preoperatively. Throughout the study the animals breathed room air. From these data the per cent of total flow through each lung was calculated, using the same principles as discussed for the acute experiments.

RESULTS

The data obtained from these experiments are listed in Tables I, II, and III. No control values for ratio of flow through each lung were obtained, but previous work^{1,4} has shown that 40 to 45 per cent of the cardiac output in the dog perfuses the left lung. In the acute experiments (Table I), ten to forty-five minutes after bronchial obstruction, the first sampling revealed a reduction in flow through the atelectatic lung to 0 to 34 per cent of the total flow. There was no significant progression when the studies were repeated on the same dog. As a result of this marked reduction of flow through the atelectatic lung, the femoral arterial saturation never fell below 88 per cent. The pulmonary resistance in the oxygen breathing lung stayed the same or fell, while that in the atelectatic lung increased markedly (Table III).

In the chronic experiments (Table II) flow to the atelectatic lung was reduced to as little as 7 per cent of the total when the first observations were made only two days after operation, and remained essentially unchanged during further observations. The systemic arterial saturation in these chronic experiments was somewhat lower than in the acute ones, ranging from 77 to 92 per cent.

DISCUSSION

The degree of decrease in flow through an atelectatic lung with widely open pleural space and while the animals received artificial respiration was of the same order of magnitude as that found by Moore¹ and Andrus.² Since no significant progression was found in ratios of blood flow through the two lungs

after the first study, ten or more minutes after bronchial ligation, this maximal effect may well have occurred as soon as the lung became collapsed. In the animals of the acute series, in which most of the nitrogen was washed out by preliminary bilateral oxygen breathing, complete collapse of the lung was evident within five minutes. A slower progression of atelectasis was observed in the chronic series where room air was breathed through the study.



Fig. 2—Dog No. 16. Intravenous angiogram taken six months after production of atelectasis. Note unchanged diameter of large vessels and failure of filling of small vessels on atelectatic side.

A second difference in the two series was evident from the levels of the arterial oxygen saturation during complete atelectasis. The somewhat higher saturations in the acute experiments were due to the relatively large amount of dissolved oxygen in the blood draining the oxygen-breathing lung. This resulted in an oxygen content of this blood well in excess of the oxygen carrying capacity of the hemoglobin.

The clinical significance of the magnitude and promptness of reduction of flow following atelectasis is evident. This adjustment prevents a significant drop in arterial saturation when parts of the lung are collapsed during intrathoracic procedures. It is similarly this reduction in flow which enables the arterial oxygen level to be sustained in chronic atelectasis.

It is of interest to point out that reduction in flow due to atelectasis was far greater than that found by us during unilateral nitrogen breathing.⁴ This strongly suggests the importance of the mechanical factor (collapse of the lung) in the former. The exact location of the increase in resistance to flow of the atelectatic lung is uncertain. Fig 2 shows that the large vessels did not appreciably decrease in diameter. The locus must, therefore, be in the small vessels of the lung.

SUMMARY

In two groups of dogs atelectasis of one lung was produced by bronchial ligation. The first group was studied up to three hours after ligation with the pleural space widely opened, under artificial respiration. The flow to the atelectatic lung was reduced to 0 to 34 per cent of the total blood flow due to a marked increase in pulmonary vascular resistance. Maximal effects were observed in the first postatelectatic study, ten to forty-five minutes after ligation.

The second group of dogs was allowed to survive for periods up to six months. Reductions in flow similar to that in the first group were observed.

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DISCUSSION

DR. J. J. FINNERTY, New York.—This work represents an excellent development of the fundamental work of Andrus, Moore, Bergrenn, and Björk. While working with Professor Crafoord, Dr. Carlens and I had an opportunity to make some observations which are pertinent to this work.

Variations in the oxygen saturation of the hemoglobin of peripheral arteriolar blood in fifteen patients undergoing thoracic operations were observed by means of a sensitive meter. Adequate ventilation was assured by the use of a mechanical positive pressure anesthesia apparatus, and a double lumened endobronchial anesthesia tube was employed, which permitted inflation or deflation of the lung on the operated side at will.

Upon entering the pleural cavity and disconnecting the lumen of the endobronchial tube going to the operated lung, thus allowing it to collapse, falls in the hemoglobin saturation of the peripheral blood varying up to 40 per cent were noted. This fall was interpreted as being due to the flow of reduced blood through the nonventilated lung, with its consequent return to the peripheral circulation in its original unsaturated condition. This fall in effect a pulmonary arteriovenous shunt.

Occlusion of the pulmonary artery to the deflated lung obliterated this 'shunt' and, as a result, the peripheral saturation immediately rose to normal or near normal levels. It was further noted that the fall in saturation was roughly inversely proportional to the degree of pathology in the operated lung, being absent or minimal in totally destroyed

lungs, and maximal in well functioning lungs, as in the case of a small peripheral tumor. This, of course, suggests that as a lung ceases to function, as in atelectasis or suppurative disease, its pulmonary blood flow decreases.

We believe that these observations are clinically significant for the following reasons: first, long periods of collapse of the operated lung that has an intact pulmonary blood flow lead not only to hypoxemia, but probably also to carbon dioxide retention and respiratory acidosis. This can be corrected either by maintaining adequate ventilation to the operated lung, which may make surgery difficult, or by early occlusion of the pulmonary artery to the operated lung. Furthermore, occasionally we see a patient with bilateral emphysema in whom, upon opening the thorax, and permitting the operated lung to deflate, there develops marked respiratory embarrassment and cyanosis, of such a degree as to make us consider abandoning the operative procedure. Early occlusion of the pulmonary artery to the operated lung, closing the "shunt" in many cases results in such marked improvement that the operative procedure may be completed uneventfully.

DR C ROLLINS HANLON, St Louis—We were interested in this problem several years ago and, in conjunction with Dr Dean Warren at Johns Hopkins University, we carried out some physiologic studies of the blood flow and other factors associated with atelectatic lungs in dogs. In our experiments the right main bronchus was divided, the proximal end sutured and the distal end left unsutured. We found, as was reported here, that immediately after this procedure there was some depression of the peripheral oxygen saturation of blood. It was usually surprisingly low, in the neighborhood of 55 or 60 per cent. We assumed that that was due to one of two things, either because there was some interference with blood flow to the good lung, due to atelectasis, or because a considerable amount of blood was still going through the atelectatic lung. As time went on, two to six months thereafter, the oxygen saturation of arterial blood gradually rose so that the standard level after two to six months was 88 to 90 per cent, indicating that relatively little blood was running through the atelectatic lung. Therefore we assumed that in the acute stage the unoperated lung was somewhat below par so far as efficiency of oxygenation was concerned. This was confirmed by the finding of patchy atelectasis in the unoperated lung at autopsies which were done electively on dogs one or two days after the operation was carried out.

With regard to pulmonary vascular resistance, we attempted to investigate this by banding the opposite pulmonary artery with reactive Cellophane, so that as months went on one would get constriction of the opposite pulmonary artery driving blood to the atelectatic lung. By this device, which is a useful experimental device for cyanosis (and that is the main reason I mention it) one can achieve, two to six months thereafter, arterial oxygen saturation varying from 65 to 79 per cent as a continuing level.

DR ROSS ROBERTSON, Vancouver, B C—As one who uses endobronchial anesthesia very extensively for pulmonary surgery, I am very grateful to Dr Peters for his paper. With endobronchial anesthesia, surgery of the lungs is a great deal easier, but we have always been worried as to whether or not the shunt from the atelectatic lung interfered with the oxygenation of the patient. In a few cases we have taken arterial oxygen saturation after the endobronchial catheter was placed, and have found that before the chest was opened there is quite a drop in oxygen saturation of arterial blood. Consequently, in using endobronchial anesthesia, there is some urgency in opening the chest, but as soon as the chest is opened and the lung can collapse, and the vessels collapse with it, the oxygen saturation rapidly rises to approximately 85 to 90 per cent, and so we feel justified in using this type of anesthesia.

DR PETERS (Closing)—Regarding this marked fall in saturation discussed by Dr Finnerty, it is important to point out that if animals are given pure oxygen, the amount of dissolved oxygen in a pulmonary vein on the aerated side represents two or three cubic centimeters per 100 cc of blood and in this manner the systemic arterial oxygen satura-

can be protected even if there is flow through the nonaerated lung. Likewise, the extent of collapse is extremely important. In the experiments in which dogs breathed nitrogen in the lung, the decrease in blood flow was much less than when the lung was collapsed. Thus if the mechanical factor is not utilized the flow will be greater and therefore the saturation lower. The low saturation might represent poor ventilation of the aerated side.

The oxygen saturation in animals which have 20 per cent of their blood flowing through an atelectatic lung will range, if air is used for breathing, down to 84 or 85 per cent. If the dog is breathing oxygen as in the acute experiments the oxygen saturation is at almost a normal level. Our oxygen saturations have been in much the same range as those spoken of by Dr. Hanlon except for the fact that compensation occurred immediately in these experiments and we did not notice any significant further decrease in flow with subsequent observations.

With regard to the use of endobronchial anesthesia, the only suggestion I would make would be to be sure that the patient has all the nitrogen washed out of the lungs and blood by oxygen breathing, so that immediate collapse of the lung will occur following bronchial clamping.

SURGICAL MANAGEMENT OF PULMONARY EMPHYSEMA

GERALD L CRENSHAW, M D (BY INVITATION), AND
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BASED upon the theory that pulmonary emphysema results from bronchospasm and avascularity of the lung with secondary nutrient degeneration, a preliminary report of a new idea in surgical management of pulmonary emphysema is presented. It is felt that the degenerative cycle of pulmonary emphysema may be interrupted by establishing collateral circulation to the lung.

From a physiologic and pathologic standpoint, pulmonary emphysema is divided into four categories: (1) diffuse hypertrophic emphysema, (2) subpleural bullae and blebs, (3) localized degeneration, more commonly known as "cotton-candy lung", and (4) a combination of two or three of these conditions.

The etiology of pulmonary emphysema has been variously attributed to chronic cough, loss of elasticity, recurrent bronchial infection, bronchospasm, and occupational hazards, or has been considered as secondary to chest wall changes^{6, 11}. However, pathologically the bronchial arteries may show arteriosclerotic and endarteritic changes which may produce loss of elasticity with thinning of the alveolar walls and degeneration. Thus there is a resultant diminution of the capillary bed of the lung,¹³ although there is some difference of opinion on this latter point.²

In spite of marked pressure differentials in the systemic and pulmonary vessels, it is well known that, in the normal lung, there is free communication between the two systems through the subpleural capillaries. In the diseased lung, it has been demonstrated, pathologically and experimentally, that there are blood shunts which allow free flow from the diseased tissue into functioning alveoli.¹⁵
^{16 17 22} These blood shunts are physiologically similar to air drifts.^{7 20 21} They develop between the systemic and pulmonary systems wherever infection in a lymph follicle takes place, and have been demonstrated to exist freely in bronchiectasis, pulmonary emphysema, and congenital pulmonary atresia.¹² Secondary to infectious processes, these vascular shunts occur near the termination of cartilaginous bronchi.

In animal experiments, by varying pressure differentials in systemic and pulmonary arteries, systemic blood has been demonstrated beyond the pulmonary capillaries. If the pressure is high enough or maintained long enough, systemic blood can be demonstrated, by catheterization studies, to be present in major segmental pulmonary arteries. This retrograde flow of systemic blood would then be shunted into other pulmonary radicles. In dogs the systemic to pulmonary

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shunt has been shown volumetrically to be one-third greater than the normal output of the left ventricle¹² Study of increased pressure within the pulmonary artery has shown retrograde flow as far as the aorta¹⁰

It has long been established that a collateral blood supply, by way of the intercostals, enters the lung through vascularized adhesions For this reason, in doing intrapleural pneumonolysis for tuberculosis, the procedure is done within a period of two months from the onset of collapse so that troublesome bleeding from vascularized adhesions can be avoided It has been noted, in tuberculous patients in whom emphysema might be expected, that under areas of pleural symphysis and vascularized adhesions emphysema actually does not occur¹⁴ This is contrary to what would be expected with the proliferative changes in the blood vessels and bronchostenosis in tuberculous lungs

In certain cases of pulmonary atresia, life has been maintained by the ingrowth of collaterals from the intercostal, internal mammary, pericardiophrenic and esophageal vessels^{4, 5, 19} These new collaterals communicate with the pulmonary artery radicles, through shunts and through the alveolar capillaries In fact, thoracotomies with pleurectomies have been performed to establish such new collaterals⁵

It has been demonstrated in animal experiments that prolonged bronchial obstruction produces irreversible emphysema Chronic partial bronchial obstruction, either by bronchospasm or secretions, is felt to be an important factor in clinical emphysema Certain clinical cases of emphysema have benefited by complete denervation (sympathetic and parasympathetic), which reduces the factor of bronchospasm¹

The benefit derived from the surgical removal of space consuming bullae and blebs has been pointed out⁹ In our opinion, this is equally true of atrophic lung

OPERATIVE TECHNIQUE AND MATERIAL

The above ideas are incorporated in a surgical approach to the treatment of pulmonary emphysema An open thoracotomy is done Degenerated lung is removed by segmental resection or lobectomy Subpleural bullae and blebs are removed by clamp resection Smaller bullae and blebs are treated by individual mattress sutures As far as possible, all air leaks are controlled Complete vagal denervation is accomplished by severance of branches at the hilum, including those from the left recurrent laryngeal nerve Perivascular and peribronchial tissues are dissected and the inferior pulmonary ligament is severed (Blades' technique¹) The sympathetic chain is removed from the third through the ninth ganglion, including the splanchnic nerves The parietal pleura is removed from the chest wall Irritative sterile talc is powdered over the visceral pleura When the chest is closed, two large intercostal tubes are inserted to which regulated suction is applied as soon as the patient is returned to his room

Eleven cases of pulmonary emphysema have been so surgically managed Table I. Ten cases have had unilateral procedures and in one case excision of the disease on the opposite side had been done three years prior to the present series

TABLE I CLINICAL RESUME OF 11 CASES OF PULMONARY EMPHYSEMA SURGICALLY MANAGED

NAME	AGE	SEX	SYMPTOMS	IMMEDIATE PREOPERATIVE STATUS	ROENTGENOGRAM DIAGNOSIS	GROSS PATHOLOGY OF SURGICAL SPECIMEN	TIME OF FOLLOW UP AFTER SURGERY	CLINICAL STATUS AT LATEST DATE OF FOLLOW UP
B G	60	M	Dyspnea, orthopnea, cyanosis, mild	Condition critical	Diffuse pulmonary emphysema, bilateral, compression	None	14 mo	Full time activity, dyspnea only on exertion, lies flat for sleeping
I K	48	M	Marked dyspnea, orthopnea, cyanosis, marked	Condition critical	Diffuse pulmonary emphysema, bilateral	None	15 mo	Working 8 hr day as counterman in lunch room, mild dyspnea on exertion, no cyanosis, lies flat for sleeping
T H	59	M	Marked dyspnea, orthopnea, cyanosis, mod	Condition critical	Bullous emphysema, bilateral	Degenerated lung, lobar and segmental	14 mo	Working full time, managing lumber yard, mild dyspnea on exertion, lies flat for sleeping
D M	38	F	Asthma, dyspnea, mod, orthopnea	Activity markedly restricted	Diffuse pulmonary emphysema, bilateral	None	10 mo	Has resumed full household duties, mild dyspnea on exertion, asthma continues
R F	65	M	Dyspnea	Ambulatory but activity becoming more restricted	Bullous emphysema, bilateral	Bullous emphysema, chronic pleuritis	8 mo	Full time activity, mild dyspnea only on exertion
I Y	60	M	Marked dyspnea, orthopnea, cyanosis, marked	Bedridden, under oxygen therapy	Bullous emphysema, bilateral	Degenerated lung, lobar and segmental	6 mo	Has returned to full time heavy labor, driving logging truck, lies flat for sleeping
A R	75	M	Marked dyspnea, orthopnea, cyanosis, mod	Activity markedly restricted	Bullous emphysema, bilateral	Bullous emphysema, degenerated lung, lobar and segmental, pleural fibrosis	4 mo	Able to walk 2 blocks, drives his car regularly on 20 mile trips, lies flat for sleeping
M T*	47	M	Marked dyspnea, orthopnea, cyanosis, marked	Condition critical	Bullous emphysema, bilateral	Bullous emphysema	2 mo	Still hospitalized, general condition improved
M S	44	M	None, disease revealed on routine roentgenogram	No disability	Bullous emphysema, bilateral	Bullous emphysema	6 wk	Working full time as office clerk
M D	48	M	Productive cough, wheezing, dyspnea on exertion	Activity becoming more restricted but able to work	Bronchiectasis left lower lobe	Bronchiectasis left lower lobe, bullous emphysema, degenerated lung, left upper lobe	5 wk	Working full time as hospital porter
I W	65	M	Marked dyspnea, orthopnea, cyanosis, mod	Completely bedridden under oxygen	Bullous emphysema, bilateral	Bullous emphysema, silicosis	14 days*	Dead, perished at air lake

Three cases were of the diffuse hypertrophic type, 2 of the "cotton-candy" type, 4 cases had bullous disease alone, and 2 cases had bullous disease with "cotton-candy lung"

The advantages of removing space consuming lesions and allowing more lung to become functional was an obvious indication for surgery. Denervation was done because of the successful experience of other surgeons in reducing bronchospasm. Parietal pleurectomy was accomplished so that the lung would become adherent to the chest wall. The ingrowth of systemic collaterals into the pulmonary tissue possibly stopped the further development of emphysema. No autopsy material is available to substantiate this point. In the cases recently operated upon poudrage has been done to enhance the pleural chest wall symphysis.

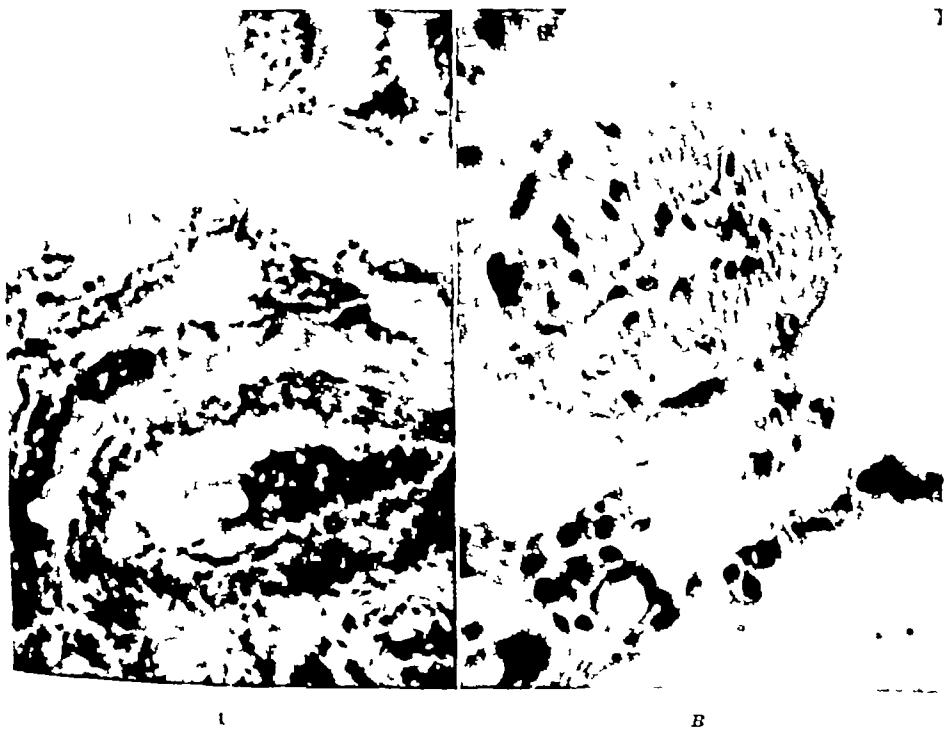


Fig. 1—A: Microscopic section of pulmonary emphysematous lung showing loss of alveoli and chronic bronchitis with peribronchovascular inflammatory changes. An associated artery is shown at the top ($\times 100$). B: Enlargement of the artery seen in A showing advanced atherosclerosis and intimal thickening ($\times 400$).

Pathologically, the surgical specimens show the typical changes of emphysema. Advanced bronchial endarteritis is demonstrated (Figs 1 A and 1 B). Chronic peribronchovascular inflammatory infiltrations are usual and frequently bronchial wall degeneration is seen.

RESULTS

Most of the patients were complete respiratory cripples in whom a medical regimen of bronchodilator drugs, oxygen, antibiotics, bronchial drainage, pneu-

moperitoneum, abdominal belts, and treatment of any concurrent *cor pulmonale* had failed to influence the progression of the disease. In the face of advanced emphysema progressing under medical management, surgical intervention was tried. With more experience, surgery was found to be indicated particularly in those patients with degenerative ("cotton-candy lung") and bullous disease.

These patients were poor risks for surgery. All were dyspneic, and some orthopneic and cyanotic. However, with the introduction of positive pressure anesthesia, their general condition moderately improved and they tolerated the operation surprisingly well. In those patients with bullous disease, as soon as the thoracic cavity was opened there was immediate improvement in their ventilation and course, as compression of the lung was relieved by the protrusion of the bullae into the wound, and this improvement continued after surgical removal of the bullae.

The immediate postoperative period in those patients with the greatest respiratory crippling was extremely stormy. It was difficult to obtain airtight closure of the lung at surgery in all cases, because of failure of sutures to hold in lung of poor tensile strength. Limitation of respiratory excursion, because of pain, embarrassed their low pulmonary reserve, and likewise interfered with adequate tussive removal of secretions. Two patients required positive pressure oxygen therapy (Bennett valve) temporarily to relieve their respiratory embarrassment. After clearing of the tracheobronchial tree at surgery subsequent bronchoscopies were not required. Early ambulation without oxygen therapy was encouraged.

The late postoperative course of three patients with bullous disease showed marked improvement. Two returned to full-time work within three months after operation. One patient with extensive bilateral bullous disease, who developed cardiac arrest on induction of anesthesia, has been followed for a period of two months postoperatively. He is now semiambulatory, but is still confined to the hospital. One death occurred, at thirteen days after operation, in a patient with extensive silicosis and bullous emphysema. Death was due to persistent air leak and partial lung collapse with ultimate respiratory failure.

The four patients who had resection of degenerated lung ("cotton-candy lung") showed slower improvement than those patients with subpleural bullae and blebs. One patient returned to limited work within seven months after operation, and within one year was doing full-time work, managing his own lumber yard. Another of these patients returned to full-time heavy labor, driving a logging truck, within four months after operation. The late postoperative course of a third patient in this group is described in Case 2. The fourth patient in this group has just returned to full-time work, as a hospital porter, after five weeks' postoperative convalescence.

The three patients with diffuse hypertrophic changes required three to four months before slow improvement was noted. One patient was able to resume her usual household duties within a period of six months after operation. Attacks of asthma following an upper respiratory infection caused a return of marked dyspnea and cyanosis, but this subsided with clearing of the infection and she is again doing all her housework. Another patient was working eight hours a day, as counterman in a lunch room, within six months after operation, and has

continued at this work with only one interruption of two weeks' duration because of a severe bronchial infection, which cleared promptly under antibiotic therapy. The late postoperative course of the third patient is described in Case 1.

CASE REPORTS

CASE 1—B G, a 64-year old white man, was first seen by us in Peralta Hospital on June 15, 1948. The referring physician desired bronchoscopy because of difficulty in breathing secondary to an acute bronchial infection. The patient, a retired manufacturer, had recently been sent to California from the east coast because of asthma and progressive dyspnea. Bronchoscopy revealed bilateral thick sputum. There was generalized edema and reddening of the tracheobronchial mucosa.



Fig. 2.—(Case B G) April 7, 1952. Postoperative roentgenogram showing narrowing of left intercostal spaces.

The medical management of this patient consisted of the following. His smoking was completely restricted. He was placed on a salt free diet, on breathing exercises, and on Aero and intramuscular antibiotics. He was given mercurial diuretics for cor pulmonale. Under this medical management he temporarily improved, but again progressive dyspnea and orthopnea were noted. Finally, in May, 1950, hospitalization was required because of exacerbation of these symptoms, and he failed to respond to diuretics, antibiotics, or to continuous oxygen therapy. Surgical management by denervation and resection of bullae had been reported, and the idea of pleurotomy, with development of collateral circulation, occurred to us. With the failure of medical management in this case, surgical intervention was thought worth trying.

Physical examination just prior to surgery revealed a dyspneic, orthopneic, and emphysematous man, whose condition was critical. Pertinent findings were an emphysematous chest with suppression of breath sounds and prolonged expiration through pursed lips. Wheezes and rhonchi were heard at both bases.

Laboratory work revealed a polycythemia of 6,200,000 red blood cells, and a hemoglobin of 110 percent. A chest roentgenogram showed typical findings of generalized hypertrophic emphysema.

On May 12, 1950, a left open thoracotomy was performed, under positive pressure anesthesia which the patient tolerated surprisingly well. A diffuse type of emphysema, without bullae or bleb formation, was found. The pulmonary artery was markedly enlarged. Blades' technique of vagal denervation to the lung, removal of sympathetic dorsal ganglion chain, severance of inferior pulmonary ligament, and perivascular and peribronchial dissection was carried out. The parietal pleura was removed from the chest wall, but not from the mediastinum and diaphragm. The chest was closed with two intercostal drainage tubes, to which regulated suction was applied as soon as the patient was returned to his room.



Fig 3—A and B (Case A. R.) Dec 17 1951. Posteroanterior and right lateral roentgenograms showing pulmonary emphysema with bullous formation at right top.

The postoperative course was stormy, due to ineffectual cough and poor respiratory reserve. Minimal pleural fluid absorbed within a few days. Within two months after operation the patient became ambulatory, and has continued to improve slowly. His blood count is normal. He now makes routine trips across the continent, and is dyspneic only on extreme exertion, consistent with his age. He was recently hospitalized for bilateral virus pneumonia, from which he made an uneventful recovery.

It is interesting to note that this patient has had episodes of bronchial wheezing during asthmatic attacks as marked on the denervated side as on the opposite side. However, physical examination usually reveals a free passage of air in the bronchial tree of the operated side, while on the opposite side multiple rhonchi and rales frequently can be heard. There has been an obvious decrease in the circumference of the operated side of the chest, and roentgenograms now reveal a decided narrowing of the intercostal spaces (Fig 2).

CASE 2—A. R., a 73 year old white male machinist, was referred to us by his attending physician on May 22, 1950, because of progressive dyspnea and weakness. He was unable to stand for longer than ten minutes, and could not walk more than 30 feet without becoming markedly short of breath. He had been well until 1945, when he began to develop the above symptoms following a severe cold.

Physical examination revealed a markedly dyspneic man, appearing much younger than his stated age. Pertinent findings were limited to an emphysematous chest, with diminished breath sounds throughout.

Laboratory work revealed a normal blood count and urinalysis. Chest roentgenograms showed bilateral emphysema with bullae at the right apex. An electrocardiogram was normal.

The following medical therapy was instituted. The patient's smoking was completely restricted. He was placed on heliotherapy, bronchial expectorants, and carbogen therapy. He was given mercurial diuretics for cor pulmonale. Under this regimen his symptoms were relieved temporarily. However, his general condition gradually grew worse, to the point of complete invalidism from further dyspnea and weakness, and surgery was deemed necessary (Figs 3, A and 3, B).

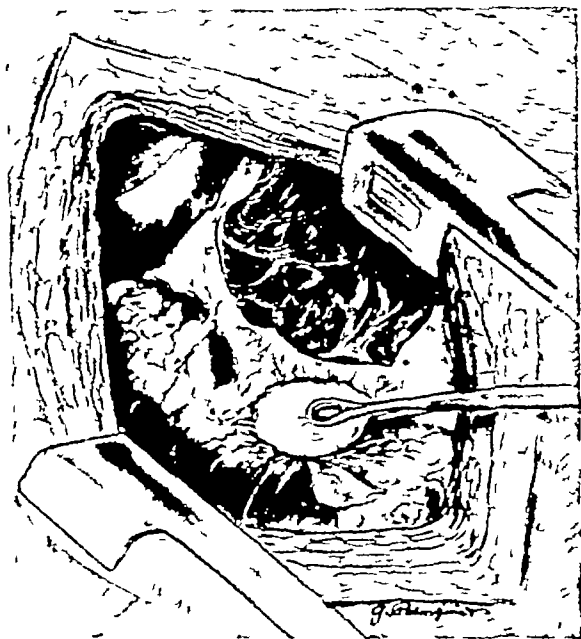


Fig. 4—Degenerated lung (cotton candy lung)

On Jan. 8, 1952, a right open thoracotomy was performed at Samuel Merritt Hospital, under positive pressure anesthesia. The entire upper lobe and the superior segment of the lower lobe were found to be degenerated (' cotton candy lung ') (Fig. 4). The remainder of the lung showed diffuse hypertrophic emphysema. A lobectomy, segmental lobectomy, and complete denervation were done. The parietal pleura was removed from the chest wall, and air leaks from raw surfaces were controlled as far as possible. Irritative sterile talc was spread over the visceral pleura, and the chest was closed with two drainage tubes, to which suction was applied as soon as the patient was returned to his room.

The postoperative course was stormy due to mild respiratory embarrassment and persistent air leaks of six days' duration. Localized fluid formed and required one aspiration. On eighth postoperative day the patient became ambulatory and was discharged home on ninth hospital day.

He has showed low but steady improvement (Figs. 5 A and 5 B), and is now able to walk 10 blocks and drives his car twenty miles without difficulty.

Pulmonary ventilatory studies, including cardiac catheterization, were done preoperatively and at three months postoperatively. Comparison of the findings of the two studies reveals no significant changes. Subsequent studies are planned.

SUMMARY AND CONCLUSIONS

Various aspects of pulmonary emphysema are discussed, and the rationale of the surgical approach to treatment is given. The object of the surgery is to allow more lung to become functional, to reduce bronchospasm, and to supply the remaining lung with collateral circulation. The manner by which this systemic collateral circulation reaches the pulmonary capillaries is described.

Eleven patients with advanced pulmonary emphysema, including the three types of diffuse hypertrophic, subpleural bullae and blebs, and degenerated lung, have been surgically managed by removal, from one lung, of degenerative and bullous disease, complete denervation, and parietal pleurectomy. Degenerated lung was found to be lobar or segmental in distribution. In all instances, the pathologic study of the surgical specimens revealed advanced changes in the blood vessels and in the bronchi.

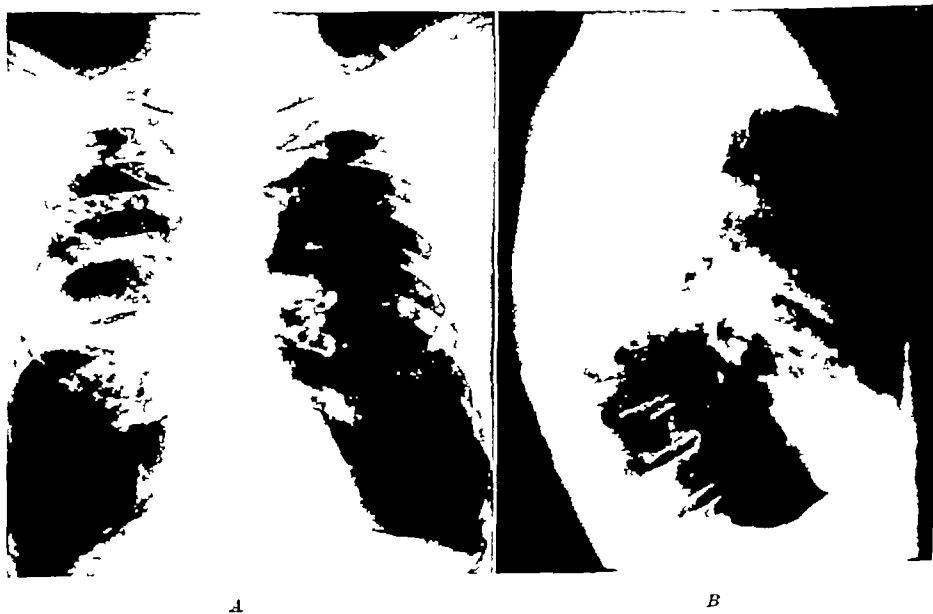


Fig 5—A and B (Case A. R.) April 8, 1952. Posteroanterior and right lateral roentgenogram showing pleural symphysis right with localized pleural fluid collection.

Although, in the main, the patients in this series were severe respiratory cripples, they tolerated the surgery well. One death occurred thirteen days postoperatively due to persistent air leaks. The immediate benefit in these patients was undoubtedly due to the denervation and removal of space-consuming lesions, but their continued improvement must be attributed to the ingrowth of collateral circulation.

TABLE II. RESULTS OF PULMONARY FUNCTION STUDIES

	PREOPERATIVE	POSTOPERATIVE (3 MO)
Cardiac output	5.5 L/min	
Heart rate	110	110
Stroke volume	32 cc	
Minute volume	7.24 liters	6.8 liters
Tidal air	575 cc	600 cc
Arterial saturation	90 per cent	89 per cent
P/Q	0.92 per cent	
Pulmonary artery pressure	37/21	
pH	7.42	7.42

The previous treatment of pulmonary emphysema has been able only to prolong its inevitable cycle. From our observations in the present series of patients it is felt that the degenerative cycle of pulmonary emphysema may be interrupted by establishing systemic collateral circulation to the lung. The clinical improvement in these patients warrants further trial of this procedure.

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DISCUSSION

DR OSLER ABBOTT, Atlanta, Ga—I want to congratulate Dr Crenshaw on his work. This has been our major investigative problem on the Chest Service at Emory University School of Medicine since 1945. We were able to present in Boston this month the results of studies on over 300 patients with pulmonary emphysema, an operative intervention register of 125, with follow up studies both clinical and physiologic.

I would like to trace a little of the history of the operative procedure we have utilized for emphysema. First, we have not purposely used the parietal pleural stripping procedure for Stage V patients. We are certainly going to give it a try, but without preponderance, and we are very grateful for that additional concept. We have used it on tetralogy of Fallot and have had beneficial results in three patients. The origin of our application of the idea was in an operating room locker room conversation in 1946, between Dr Willis Potts, Mr Brock, and myself. They commented upon the work of a French surgeon who had suggested that parietal pleural excision should be beneficial and should be done on the next tetralogy that presented no pulmonary arteries. The discussion included a considerable amount of reference to a conversation between Dr Potts and Dr Blalock in which they both were of the opinion that this might be a valuable procedure. This is the historical background so far as I know, of this part of the problem. So far as I know, the plan of combining an upper dorsal sympathectomy with parietal pleural stripping was original with us in 1946. Our patients on whom this procedure was successful have had a marked decrease in cyanosis, full cessation of squatting, and activity potential has approached normal limits.

The next part of the problem was in 1945 when we did our first total complete plexectomy on a patient with cor pulmonale, with benefit. He died in three years and this discouraged us. Brian Blades awakened our interest again by describing complete plexectomy. Dr Samson made a major contribution in regard to the bullous aspect of the disease. At the 1949 meeting of this Association we presented the proposition that denervation procedures, plus excisional surgery, were indicated in pulmonary emphysema. We have now come to the point that we feel as follows: there are two fundamental types of disease in medicine. This is the disease which is due to a single etiologic agent, such as pneumococcal pneumonia. Then, there is another concept which we derived from a luncheon meeting of Dr Graham's residents two years ago, namely, that there is a synergism, a combination of factors involving varying agents, which goes to produce more complicated medical disease syndromes. A subdivision of this group is, that in some of these diseases the early local organic damage resultant to the disease becomes an additional factor in the progression of the disease. This is the philosophy and the fact, and a greater appreciation of this should aid in the approach to this problem and other chronic atrophic disease. I would like to present a couple of slides.

(Slide) This part of the study is where patients were evaluated with regard to the degree of bronchospasm present in the emphysematous individual. Only 85 per cent of our series of 250 had demonstrable bronchospasm by our present methods. This is one of the types of measurement of maximum breathing capacity before and after the injection of many different drugs. Usually these tests are described by the patient when he tells you which drug is most effective.

(Slide) This represents an improved technique for transacting emphysematous lung tissue which we devised because we had tragic experiences in 1946 and 1947 of patients with diffuse hypertrophic emphysema, dying because of interstitial hemorrhage or hemorrhagic pneumonitis arising from the course of the suture line. The principle that we should suture through nonflowing blood is applied here. The Potts' coarctation clamp

(Slide) This represents the preoperative film. This is a centrally functioning lung even at the 1 ft bar, hypertrrophic emphysema and I am fascinated to see that we have the same pattern in these arteries that Dr. Peters showed with the obstruction in the left lobe. The postoperative film shows a functionalization of a previously compressed left lung.

(Slide) These show the various types of emphysema which may be cured or palliated. The premise, therefore, is that all types and combinations of pulmonary dysfunction are potentially curable in their earlier stages. Certainly we have shown that oxygenation is possible even in the fifth or last stage of the disease.

DR. FAX O. CARTER (New Haven Conn.) If I may be permitted I should like first to congratulate Dr. Croushaw for his interest in emphysema because I think this is one of the single most important pulmonary diseases which we see. Second I should like to allow the society to take issue with his logic. We have been concerned with emphysema for several years and the more we study it the less we know about it. But it has come to me that definitions are of great importance. When one speaks of hypertrrophic pulmonary emphysema he must exclude asthma or bronchospasm and that can be excluded in the laboratory of course. Although the characteristic hypertrrophic emphysema may follow a long course of asthma certainly we must distinguish the surgical objectives in the operation aimed at asthma by nerve cutting methods or is it aimed at emphysema which is, by definition, is not asthma? Second there is the problem of pulmonary cysts. It is true that diffuse bullae and blebs are present in many patients with emphysema, but there are other causes for pulmonary cysts as well. These may impair function by expansion of their own size, thereby compressing adjacent pulmonary tissue and interfering with ventilation. Third, is the type of emphysema you see in inflammatory states. Certainly, patients with fibrotic contracted fibrotic tuberculous lobes will have overdistention of adjacent lobes and that, to be sure, is emphysema. But certainly etiologically and pathologically it must be distinguished from those patients with bronchial obstruction which function is called hypertrrophic emphysema.

What is this hypertrrophic emphysema? Long ago Drs. Anderson and Spink described bronchial obstruction of a hyaline nature in patients with this far advanced disease. It is difficult, so that all lobes are symmetrically enlarged. But in the disease we may have isolated blebs and bullae. We have found, as have others, that these patients do not have reduced ventilatory function to the point of a maximum breathing volume of perhaps 2.0 or even 1.5 liters per minute will have an apparently increased residual volume. That is to say, a drop of 10 liters due to bronchospasm is more important when you have a maximum function of only 2.5. I am impressed by the fact that this is truly, therefore ventilatory impairment. The problem with these patients is that they cannot get enough air in and out of their lung. A conductance problem is the

increased residual air, which means that every breath that comes in is diluted in a large volume, much greater than the normal. In other words, residual air in the normal is approximately 20 per cent of full lung volume and, in emphysema patients, it may become as large as 50 or 60 per cent of total lung volume. There is, therefore, retention of carbon dioxide and there may actually be a change in the blood pH in advanced cases. Conversely, the oxygen tension at the alveolo capillary membrane is markedly reduced.

If it is not possible for an individual to get enough air in and out of his lungs, you do not make him any better by bringing more blood to his lungs. And these patients are therefore not comparable to the tetralogies or other blue cardiacs because in those cases there is ample air in and out of the lung but there is not adequate blood, or at least the blood is not going in the right place.

The reasoning therefore seems to me fallacious that one can improve emphysema patients of the hypertrophic type by pleural poudrage.

The last point I would like to make is that almost regardless of what you do for these chronic invalids, if you do it carefully, with tender loving devotion, they get temporarily better. If you put them in a hospital and increase their bronchial drainage with ammonium chloride, if you give them a few whiffs of oxygen with a pressure apparatus, if you give them a large volume of antibiotics, they will be improved, and one must exclude that improvement from the improvement specifically related to the surgical operation. Dr Whittenberger and others have demonstrated that if you put such a patient in a respirator so that his residual volume is reduced to normal mechanically, a few hours a day for a week or so, he will have improvement, some of which remains even for a year. We have shown similarly, that if we give these patients pneumoperitoneum and if it is efficient, functionally effective and tolerated well, it may be maintained for perhaps six months and then abandoned and the patient will maintain a certain percentage of that improvement for up to two years. I would like to suggest, therefore, in summary, that the improvement which is being attributed to the increased vascularity of the lung is, in effect, due to a combination of many factors, some of which may partially reduce the residual air, some of which improve the bronchial toilet, diminish the secretions and make the ventilatory function of the lung somewhat more effective.

DR CRENSHAW (Closing).—I anticipated Dr Carter's criticism and, in answer, I would like to reiterate that it is our feeling that bronchospasm undoubtedly plays a large part in the production of all three types of emphysema, but it is hard for me to conceive that bronchospasm alone can cause such massive destruction of a lobe or segment as seen in these pictures.

Before reporting our theory and procedure we have waited for an observation period of approximately two years on some of our patients. I would like to call attention to the fact that the patient followed for the longest period of time had failed to respond to a medical regimen, as outlined by Dr Carter, and therefore surgery was decided upon. This patient was of the diffuse hypertrophic type. Since surgery he has required no medical regimen for the emphysema. He recently went through an attack of bilateral virus pneumonia with rapid recovery. He is now able to make trips across the continent and leads a normal life for a man 67 years of age.

I want to thank both Dr Abbott and Dr Carter for their comments.

OBSTRUCTIVE PNEUMONITIS SECONDARY TO BRONCHIAL ADENOMA

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BRONCHIAL adenoma is usually a slow-growing tumor which metastasizes in about 5 to 10 per cent of cases but which, by bronchial occlusion and its effects, often does its most serious harm to the patient. This latter aspect of these tumors has been often mentioned in the literature and is familiar to those who deal with such problems. However, several questions arise in regard to suppurative disease associated with adenoma. Such questions as to frequency, form, and effect on resectability, morbidity, and mortality are those to which an exact answer based on a large experience is not sufficiently emphasized in the literature. This study was undertaken in an attempt to answer some of these questions and to emphasize other known facts.

HISTORICAL

In Mueller's¹ case in 1882, which was the first case of bronchial adenoma described, many bronchiectatic cavities distal to the tumor were found at necropsy. In Heine's² case in 1927, bronchiectasis also was distal to the tumor.

Rusner,³ in 1928, reported a case in which death was due to putrid empyema, and an obstructing bronchial adenoma was found at necropsy. Kramer,⁴ in 1930, reported two cases in which multiple attacks of pneumonia and pleurisy occurred. Wessler and Rabin,⁵ in 1932, reported seventeen cases of benign bronchial tumors, twelve of which were adenomas. From a study of the reports of these cases, it seems apparent that in at least eight of these twelve there was evidence of moderate to severe obstructive pneumonitis.

Foster Carter,⁶ in analyzing the clinical features of all cases reported up to 1941, including twenty-two of his own, found that in 38 per cent of all cases there was clinical evidence of pleurisy and pneumonia, in 15 per cent there was bronchiectasis, in 7 per cent there was empyema, and in 2 per cent there was pulmonary abscess. Foster Carter also analyzed the causes of deaths in his and prior cases and found that of the seventeen deaths six were due to empyema and pulmonary abscess, one was due to pneumonia, three were due to hemoptysis, three followed thoracotomy, and four were due to unrelated disease. He further stated that eventually in 60 per cent of cases bronchiectasis, pulmonary abscess, or empyema will develop. Mallory⁷ stated that one must expect bronchial pneumonia, with obstruction and destruction of pulmonary tissue in almost every case in which treatment is not given.

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Brunn and Goldman⁸ pointed out that occurrence of a lesion of the central nervous system in a case of long-standing adenoma is almost always the result of metastatic septic emboli from the associated pulmonary suppuration. They also stated that in cases of bronchogenic carcinoma episodes of pulmonary suppuration are usually continuous and progressive, and in cases of adenoma they are intermittent, and when death occurs in adenoma it is usually a result of suppurative disease.

Adams, Steiner, and Bloch,⁹ in reporting on five cases in 1942, found evidence of metastasis of malignant tissue in three of the five. However, in three of the four deaths in the five cases, suppurative pulmonary disease was the cause of death or was a major contributing factor, and in none was metastasis of malignant tissue or carcinomatosis a cause of death.

Clerf and Bucher¹⁰ found suppurative disease or atelectasis in 60 per cent of their cases. They stated that endoscopic removal of the endobronchial portion of the tumor will relieve to a large extent the suppuration, but the bronchiectasis persists. Jackson, Konzelmann, and Norris¹¹ stated that endoscopic drainage at least is always advisable in such cases. Holley¹² found chronic pneumonitis in eighteen of thirty-nine cases and "suppuration in only a few." Van Hazel, Holinger, and Jensik¹³ had one case in their series in which pleural reaction and suppuration made it impossible to perform resection.

Goldman and Conner¹⁴ found significant severe pulmonary suppuration in ten of thirty-eight cases. This was the direct cause of death in two cases in which embolic brain abscesses were present. Septic cerebral emboli occurred in another case. Two of their patients were treated by preliminary drainage (rib resection) before pulmonary resection was attempted.

Delarue¹⁵ compared the postoperative morbidity in eleven cases of bronchial adenoma in which pneumonectomy was performed with a comparable group of bronchogenic carcinoma and found 45.4 per cent morbidity in the adenoma cases compared with 24.1 per cent morbidity in the cases of carcinoma. His belief was that this was due to the associated suppurative disease found with bronchial adenoma.

RESULTS OF THE STUDY

This study is of the 102^o cases of bronchial adenoma encountered at the Mayo Clinic through December, 1950. This includes both the carcinoid and cylindroma types, and excludes any similar neoplasms of the trachea. The tissue was reviewed in all cases so that no errors in pathologic diagnosis would be included. Eighty-six of these cases have previously been reported by Moersch and McDonald,¹⁶ and some of the present data merely supplement their work.

Age—The average age was 42 years. The oldest patient was aged 70 years, the youngest 15 years. The majority of patients were in the 30-through-49-year age groups.

Sex—There were fifty-five males and forty-seven females.

Duration of Symptoms—Six patients were completely asymptomatic. Twelve patients had symptoms of nine years' duration or more (two of whom

*This group does not include an additional four cases in which the histologic pattern was not absolutely typical of adenoma, but which probably represent metastasizing adenomas. These cases plus others form the basis for a separate report.

and symptoms for thirty-six years) Excluding these twelve long exceptions the average duration was twenty-nine months Including the long exceptions, the average duration was fifty-four months

Symptoms—In decreasing order of frequency the more frequent symptoms were (1) cough (2) hemoptysis (3) recurrent episodes of pneumonitis (4) fever (5) fever (6) dyspnea

Physical Examination—In decreasing order of frequency the more frequent findings were (1) decreased breath sounds over involved area (2) dullness over affected area, (3) negative thoracic findings (twenty-six cases), (4) lag in chest movement on breathing and (5) râles over affected area In three cases a diagnosis of postpneumonic empyema had been made before the patient came to the clinic and rib resection drainage had been done in two of them

Radiographic Findings—Findings in decreasing order of frequency were (1) segmental consolidation or atelectasis (2) peripheral mass (often well circumscribed) (3) hilar mass (4) evidence of bronchiectasis, (5) negative thoracic roentgenogram (ten cases)

Bronchoscopy—Results of bronchoscopic examination were (1) negative in ten cases (seven of these peripheral location) (2) not done in four cases (peripheral location) (3) positive in 92 per cent of those examined or 88 per cent of total group *

Treatment—The 102 cases may be divided into two groups from the standpoint of treatment the nonresected and resected groups. The nonresected group consists of thirty-nine cases in which the treatment was as noted in Table I

TABLE I. TREATMENT IN NONRESECTED GROUP OF BRONCHIAL ADENOMAS

TREATMENT	CASES
Carcinoma removed alone	11
Radon treatment alone	7
Rib resection for empyema followed by diathermy	1
Expiratory nonresectable (one of these treated with diathermy and radon later)	2
Diathermy and radon seeds	4
Diathermy and roentgen treatment	2
Diathermy radon seeds and roentgen treatment	1
Treatment not advised or advised but not used	5
Total	39

No patient has been treated definitively by bronchoscopic means alone since April 1948. This is an eloquent expression of the change in opinion of the majority of the group in regard to the treatment of choice in this condition.

Factors for Pulmonary Suppuration in the Nonresected Group—Such as in this group clinical only (except for cases in which necropsy was performed) is not as reliable as in the resected group in which specimens are available for study. Roentgenographic evidence of severe sacular bronchopulmonary abscess or evidence on bronchoscopy of copious pus draining from regions distal to the tumor, or both form the evidence on which it is based.

* It is noted that tumor mass was seen or removed by obtained or both.

TABLE II PULMONARY SUPPURATION IN NONRESECTED GROUP

DEGREE OF SUPPURATION	CASES	
	NUMBER	PER CENT
None to slight	18	46
Moderate	12 }	54
Severe	9 }	

Results of treatment in the nonresected group (Table III) are essentially those described by Moersch and McDonald¹⁶ previously. Exclusive of those known to be dead, all except four of the patients in this group were followed four to seventeen years. Twenty-one of the living have been followed five years or more.

Analysis of the causes of death in the ten cases in Table III is very revealing. Seven patients died as a direct result of pulmonary suppuration, or pulmonary suppuration was a major contributing factor in the cause of death. One patient died as a result of a "heart attack," and cause of death is unknown in the remaining two cases.

TABLE III RESULTS IN THE NONRESECTED GROUP OF BRONCHIAL ADENOMAS

RESULT	PATIENTS
Living at time of last follow up	25*
Dead	10
Followed less than a year and no recent information	4
Total	39

*Twenty-one followed five years or more, four followed four years.

Resected Group—The evidence of obstructive pneumonitis in this group is more exact than in the previous group, since the resected specimens were examined in all sixty-three cases. Pulmonary suppuration was none to slight (no changes to airless consolidation without bronchiectasis or any apparent permanent change) in 42 per cent and moderate to severe (bronchiectasis, empyema, abscess, bronchopleural fistula, and so forth) in 58 per cent.

Findings at operation and their effects on ease of procedure are summarized in Table IV. Pneumonectomy was performed in thirty-two cases, and lobectomy or multiple lobectomy in thirty-one cases.

In addition there were two cases in the nonresected group in which exploration, but not resection, was performed, one because of difficulties imposed by

TABLE IV FINDINGS AT OPERATION AND THEIR EFFECT ON EASE OF RESECTION*

	CASES
No special difficulty†	30
Moderate difficulty‡	14
Great difficulty§	19

*Operation in this group done by Dr S W Harrington, Dr O T Clagett, Dr H H Gray, and Dr W D Seybold. Information for this table taken from the surgeons' notes.

†(1) Filmy adhesions or none (2) no large inflammatory nodes (3) minimal hilar induration

‡(1) Dense adhesions (2) inflammatory nodes (3) hilar induration

§(1) Empyema or pulmonary abscess (2) thick organized rind of pleura extremely difficult to dissect, no pleural space (3) marked fixation and induration of hilus, no landmarks, vessels hard to dissect and to identify

suppurative disease, and the other because of the mistaken impression that the extensive hilar induration represented a malignant lesion

Postoperative Complications—There were ten cases in which postoperative empyema developed. In eight of these ten cases the degree of suppuration in the resected lung and pleural space was moderate to severe. Four of the ten were due to leakage from the bronchial stump with fistula, and in six no definite evidence of fistula was obtained.

In seven of the ten cases hemolytic streptococcus or *Staphylococcus aureus* was obtained. *Escherichia coli* was found in one of the seven cases, and in three cases cultures were negative.

Postoperative Deaths—There were four postoperative deaths and one death which occurred during a secondary operation eleven months later for empyema. The data on these deaths are as follows:

- 1 *Right pneumonectomy* Death five months after operation due to empyema. (No suppuration in resected specimen.)
- 2 *Right pneumonectomy* Death at operation because of shock, hemorrhage, and asphyxia. (Very difficult, much suppuration.)
- 3 *Left pneumonectomy* Death third day after operation. Collapse of opposite lung. (Slight pneumonitis in resected lung.)
- 4 *Right pneumonectomy* Empyema and bronchopleural fistula. Death eleven months later of cardiac arrest during rib resection. (Moderate pneumonitis.)
- 5 *Right middle and lower lobectomy* Death due to asphyxia at operation (hemorrhage into tracheobronchial tree). (Moderate pneumonitis very difficult resection.)

Relation of Degree of Obstruction to Degree of Pneumonitis—In twenty-one of the sixty-three cases the specimens were sufficiently intact so that reconstruction of the tumor in relation to the bronchial tree could be made. In all the twenty-one, symptoms had been present from one and one-half to fifteen years. Such an examination of fixed specimens cannot of course be totally applicable to the living lung, but in general in specimens with severe degrees of obstruction the degree of pneumonitis was more severe than in specimens with lesser degrees of obstruction. In eighteen cases the obstruction was 50 to 100 per cent and pneumonitis was moderate to severe in all but one. In three cases obstruction was less than 50 per cent and pneumonitis was minimal.

There were twelve cases in which the lesions were located peripherally or were arising from a branch of a secondary bronchus only. These fit the description of the five cases described by Maier and Fischer,¹ and in all twelve the symptoms were minimal, the tumor was not visible bronchoscopically, and pneumonitis was minimal.

In general, in cases with symptoms for more than a year, suppuration was more frequent and usually more severe than in cases with shorter duration of symptoms.

Results in the Resected Group—Exclusive of the three late deaths and the four postoperative deaths, all patients except five were followed one year or

TABLE V RESULTS OF TREATMENT OF PATIENTS WHO UNDERWENT RESECTION OF BRONCHIAL ADENOMA

RESULT	PATIENTS
Living at time of last follow up	50
Dead (including postoperative deaths)	8
Insufficient follow up	5
Total	63

more Results (Table V) indicate that once diseased pulmonary tissue is removed and the patient recovers from operation there is little danger of trouble from suppurative disease

Two of the late deaths were due to metastatic malignant lesions, and the cause of death is unknown in the third case

COMMENT AND CONCLUSIONS

In recent years there has been much discussion as to the degree of malignancy of bronchial adenoma. It has been mentioned, but not sufficiently emphasized, that far more patients with bronchial adenoma have died as a result of suppurative pulmonary disease than have died from metastasis of the adenoma.

Although the antibiotics have made it possible in recent years to control infection and pneumonitis in some degree, their value may be offset by delay in diagnosis of obstructing tumor of the bronchus, if temporary resolution of obstructive pneumonitis occurs on antibiotic therapy, and the physician is not wary of the possibility of obstructing tumor. Our experience has been that, while the antibiotics will control many of the septic manifestations of pneumonitis, they will not prevent the changes in the lung which result from bronchial obstruction.

From the experience at hand it is apparent that approximately 55 to 60 per cent of patients with bronchial adenoma will have significant to severe degrees of obstructive pneumonitis. About 10 per cent of bronchial adenomas will be located peripherally and will produce few or no changes of pneumonitis.

The degree of obstructive pneumonitis appears to be directly related to the duration and degree of bronchial obstruction. The genesis and pathologic characteristics of the changes in bronchial obstruction have been described by McDonald, Harrington, and Clagett,¹⁸ and also by other earlier authors, and these descriptions need no further explanation.

It is of interest to note and compare the results of McDonald, Harrington, and Clagett's study with the present one. If one considers then grade 3 and 4 pneumonitis as comparable to moderate and severe changes, then with their adenoma series some 63 per cent had moderate to severe changes and 37 per cent had no to slight changes. With the squamous cell carcinoma group, 50 per cent had moderate to severe changes and 50 per cent had no to slight changes. In the large cell carcinoma group 16 per cent had moderate to severe changes and 84 per cent had no to slight changes. In the adenocarcinoma group 21 per cent had moderate to severe changes and 79 per cent had no to slight changes. In the small cell group 27 per cent had moderate to severe changes and 73 per cent had no to slight changes.

These figures are significant in proving the point that the longer the bronchial obstruction is present and the more the degree of obstruction, then the greater the degree of pneumonitis and suppuration. Thus, with the relatively benign (or low-grade malignant) adenoma the incidence of pneumonitis and suppuration is highest, for these patients live longer, have obstruction longer, and obstruction is usually in a centrally located bronchus. Next highest is the squamous cell group, and the incidence becomes lower the higher the grade of malignancy or the more peripherally the tumor is located.

There seems adequate basis for our belief that surgical resection of the adenomas with severe pneumonitis is much more difficult than resection of adenomas without severe pneumonitis. Actually some of the cases included in this study have been the most difficult of any pulmonary resections. In those cases with marked changes there is extreme difficulty in getting through the dense pleura. The lung may be just a mass of vessels and bronchi with marked



Fig. 1—Resected lung, which had multiple abscesses, dense pleura, and no aerated lung tissue present, all secondary to obstruction by adenoma in the main bronchus.

destruction and inflammation (Figs. 1 and 2). Edema and induration are often of such degree as to make identification and ligation of major vessels very hazardous and difficult. More than half of the resections in the series were difficult because of such reasons.

One extremely interesting observation has been that many of the patients who had severe suppurative lung disease were essentially at times afebrile, many were ambulatory, and some were even working. In this same line, although in several cases large amounts of pus were spilled into the pleural space at operation, not only did empyema not occur, but the patients were afebrile and made excellent recoveries. We are unable to explain these observations, and unfortunately do not have exact enough data to analyze such cases. In several

cases, however, the pus was found to be bacteriologically sterile, and in others apparently the body had immunized or adapted itself to the presence of such a large necrotic focus. The fact that many of the patients had received antibiotics also undoubtedly played a part in their well-being even when severe pneumonitis was present.

There was a fairly high incidence of postoperative empyema (15 per cent). We believe that obstructive pneumonitis, by increasing the technical difficulties of resection and by the presence of pus, plays a large part in the high incidence of postoperative empyema.

If one considers, however, only those cases in which operation has been performed from 1945 through 1950, the incidence of empyema is only 8 per cent. This compares with an incidence of approximately 11 per cent for empyema following lobectomy and pneumonectomy for malignant pulmonary disease (carcinoma and miscellaneous malignant lesions exclusive of adenoma).*

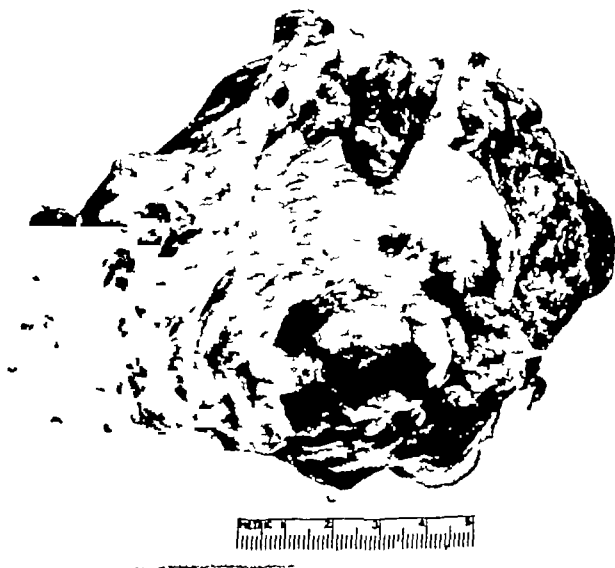


Fig. 2—Large bronchiectatic cavities in resected lung. These are distal and secondary to bronchial obstruction by an adenoma in the main bronchus.

It is believed that this lower incidence in the case of adenoma in recent years is due not so much to antibiotic and technical changes as to the fact that these patients are now undergoing pulmonary resection early before the severer changes of suppuration have occurred.

Relief of bronchial obstruction by endobronchial resection drainage may be a valuable preoperative procedure in some cases. This has been done in a few of our cases, but not in enough to enable one to draw any conclusions as to its benefit.

*Records of all pulmonary resections for malignant pulmonary disease done at the Mayo Clinic from 1915 through 1950 were reviewed to obtain this figure.

Relief of bronchial obstruction by endoscopic means did, in many cases in the nonresected group, cause relief of symptoms and apparent healing in some cases of severe suppurative disease. However, with recurrence of obstruction severe pneumonitis recurs.

The causes of death in bronchial adenoma also emphasize the importance of the suppurative factor. Of the total group, two patients died apparently as a result of multiple metastatic growths from bronchial adenoma. Exclusive of the postoperative deaths, of the other patients on whom we have adequate information seven died of pulmonary suppuration and its effects, none of hemorrhage, and one of unrelated causes.

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ADENOCARCINOMA DEVELOPING IN A PERIPHERAL BRONCHIAL ADENOMA

REPORT OF A CASE

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ALTHOUGH there is no longer doubt concerning the malignant potentialities of so-called bronchial adenomas, no well-authenticated case of bronchial adenoma showing clear-cut histologic evidence of transition to adenocarcinoma has been recorded. The purpose of this report is to describe and illustrate a case which showed convincing proof of adenocarcinoma developing in a peripheral bronchial adenoma. This observation is of considerable importance in relation to the therapy of patients with bronchial adenoma. It adds further weight to the opinion of those who believe that such lesions should be managed by conservative pulmonary resection, and that endoscopic removal is not an acceptable method of treatment.

Bronchial adenomas may be locally invasive or may metastasize by way of the lymphatics or blood stream.¹⁻⁵ Metastases to the liver, vertebral bodies, opposite lung, and kidney have been reported.^{1-3,5} In most of these cases the metastatic lesions have been reported as being similar in histologic appearance to the primary tumors, which in turn did not demonstrate microscopical features which would indicate malignant transformation. Graham and Womack⁶ expressed the belief that bronchial adenomas may lose their original features and develop into typical bronchogenic carcinomas. They reported a carcinosarcoma of the lung which they believed arose from a bronchial adenoma, but presented no convincing histologic proof for such an interpretation. Holley³ stated that a case of bronchogenic carcinoma studied at the Army Institute of Pathology presented changes which suggested a histologic transition from bronchial adenoma to bronchogenic carcinoma, but noted that the available material was not conclusive. Clerf and Bucher⁷ reported a case of bronchial adenoma in which tissue from the bronchus was examined histologically at intervals over a five year period at the end of which time carcinomatous change was detected. That finding was confirmed at autopsy. They did not state the histologic type of carcinoma which had developed.

We have seen one or two cases of undifferentiated cell bronchogenic carcinoma which had some features of bronchial adenomas, but the findings were not conclusive. In the case detailed below there seems to be unequivocal evidence of a transition from a carcinoid type of bronchial adenoma to adenocarcinoma.

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The opinions and/or assertions contained herein are those of the authors and are not necessarily to be construed as official or reflecting the views of the Navy Department or the naval service at large.

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It has been generally accepted that bronchial adenomas are invariably hilar in location. However, Maier and Fischer⁵ reported five cases arising from small bronchi with one which was peripherally located. The case described below was also of this rare peripheral variety.

CASE REPORT

Clinical History—D. L. N., a 32 year old white man, was first studied in 1945 because of the discovery, on routine chest roentgenogram, of an oval x ray density in the upper right lung (Fig 1). All physical and laboratory examinations were within normal limits. Bacteriologic studies of the sputum for tubercle bacilli were negative. He was asymptomatic and afebrile throughout four months of hospitalization. His discharge diagnosis was arrested pulmonary tuberculosis. Subsequent chest roentgenograms taken at six month intervals showed no significant change until July, 1951, when the mass was found to have enlarged markedly (Fig 2). The roentgenograms showed the lesion to be well circumscribed, dense, but not homogeneous, and several areas suggested calcium deposition as well as cavitation.

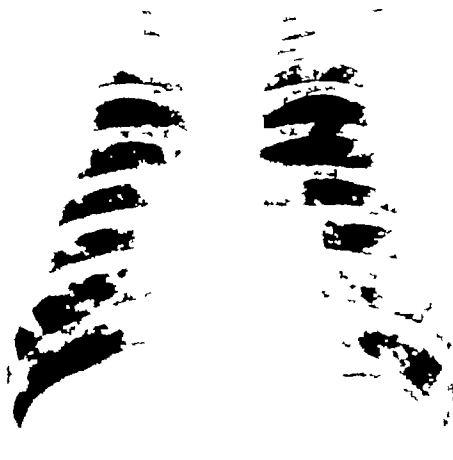


Fig 1

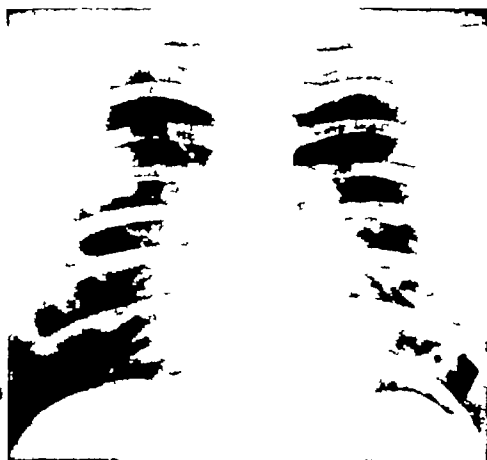


Fig 2

Fig 1.—Roentgenogram taken in 1945 showing the lesion in the peripheral portion of the right upper lung.

Fig 2.—Roentgenogram taken in 1951 showing the marked increase in size with central cavitation.

He was admitted to the St. Albans Naval Hospital on Nov. 1, 1951. At that time he was still asymptomatic and afebrile and he remained so during two and one-half months of hospitalization. There was no cough, chest pain, hemoptysis or sputum production. Breath sounds were slightly diminished, and scattered dry rales were audible in the right paravertebral area below the inferior angle of the right scapula. Second strength tuberculin skin test was slightly positive. Bronchoscopic examination revealed no positive findings and aspirated bronchial secretions were negative for acid fast bacilli and tumor cells.

On Jan. 3, 1952, right thoracotomy was performed. A rounded, moderately firm mass, 4 centimeters in diameter was encountered in the posterior segment of the right upper lobe, but not attached to, the pleura. Frozen sections were reported as "well differentiated adenocarcinoma" and a right upper lobectomy was performed. The hilar nodes were grossly enlarged. Several were removed for histologic study. The patient's postoperative course was uneventful.

Pathologic Examination—

Gross—The tumor consisted of a lobulated, firm, necrotic, spherical mass 5.0 by 6.0 centimeters. Its borders were fairly sharply demarcated but nonencapsulated. The cut surface showed a massive necrotic center surrounded by soft, friable, grayish

tissue. The adjacent pulmonary parenchyma was slightly firmer than normal, and its cut surface was grayish to yellow brown. Three lymph nodes showed only anthracotic pigmentation.

Microscopic. The tumor pattern varied considerably in different sections and within portions of the same section. It was largely medullary or alveolar with masses of closely packed, small, cuboidal cells with clear cytoplasm and small, round, vesicular nuclei. Small glands or tubules were formed in a few places (Figs 3 and 4). Stroma was sparse in these regions, and the general appearance was like that of a carcinoid tumor. Elsewhere the cells were slightly larger with eosinophilic or clear mucous-cell type cytoplasm (Fig 5). Con

Fig 3

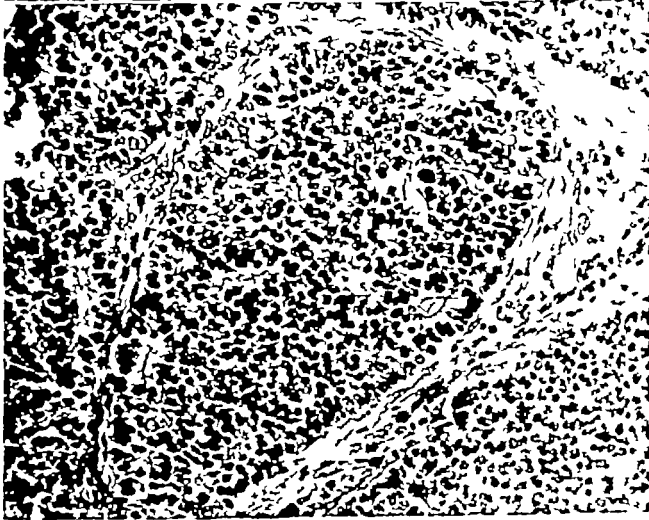


Fig 4

Fig 3—Typical histologic appearance of a bronchial adenoma. A few small glands are seen. Note the stroma separating the bronchial epithelium from the tumor. ($\times 50$)

Fig 4—Same area under high magnification. Note the strong resemblance to carcinoid tumors. ($\times 125$)

trating sharply with this appearance was that of other portions of the tumor which presented malignant appearing glandular structures separated by varying amounts of dense fibrous connective tissue (Fig 6). These glands varied markedly in size and shape and were lined by cuboidal or columnar cells with hyperchromatic nuclei showing considerable anisokaryosis and frequent mitoses (Fig 7). The benign portion of the tumor appeared to be arising multicentrically from small bronchi or bronchioles. In many places the normal bronchial epithelium could be seen to end abruptly and to widen out into bands of tumor (Fig 8). Foci of squamous metaplasia were numerous in bronchi within the tumor (Fig 9). Several lymph nodes were negative for tumor.

Fig 5



Fig 6

Fig 5 - Area showing tall mucous type epithelium lining a glandular space and filling it in places. (x100)
 Fig 6 - Area of densely cellular malignant appearing tissue. Note the desmoplastic reaction.

DISCUSSION

This case presents several interesting features. The first is the clinical course. The roentgenographic appearance was so suggestive of pulmonary tuberculosis that that diagnosis was still entertained after thorough and prolonged study, despite the absence of symptoms or bacteriologic confirmation. When, six years after its discovery, the lesion suddenly enlarged and developed central cavitation, it was interpreted as indicating extension of a tuberculous process with central necrosis. It seems highly probable that the adenocarcinoma

Fig 7

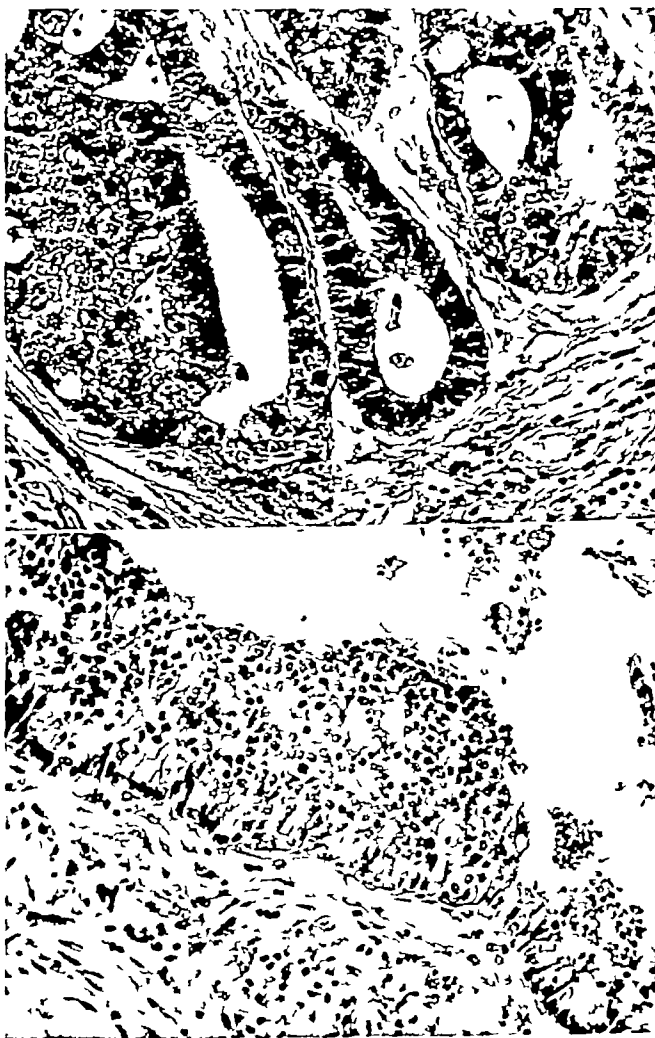


Fig 8

Fig 7—Same area under high magnification. Compare with benign appearance of Fig 4 ($\times 125$)

Fig 8—Bronchial wall showing junction of normal epithelium and neoplastic tissue which appears to be arising from the lining mucosa. ($\times 125$)

was not present initially, and developed in the adenoma at the time of the rapid increase in size of the tumor. This case is remarkably similar to that reported by Ueki and Bucher¹ which underwent malignant change five years after its detection.

The secondary pulmonary changes usually seen in bronchial adenomas were minimal due to the fact that the tumor was peripherally located, and therefore did not obstruct a large bronchus. The findings in this case agree with those of Anderson² who stated that when the diameter of a bronchial adenoma exceeds five centimeters it has been present for at least eight years (as determined from the date of onset of symptoms). The tumor in this case measured six centimeters in its largest diameter and was known to be present at least six years, and probably considerably longer.

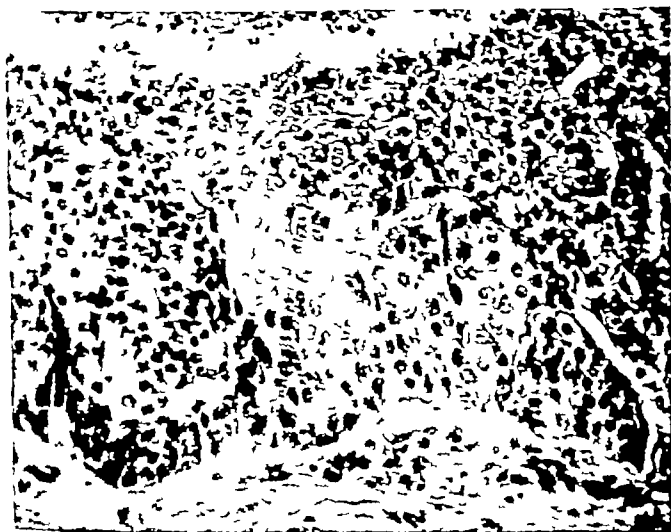


Fig. 1.—Bronchial wall involved by neoplasm and showing an area of squamous metaplasia. ($\times 125$)

In addition to its unusual location and histologic combination, this neoplasm presented still another remarkable feature that of its apparent origin from bronchial epithelium. This is contrary to most concepts which favor origin from bronchial ducts,¹¹ bronchial glands,¹¹ ducts or glands,¹² oncoocytes,¹³ undifferentiated bronchial buds.¹⁴ The bronchial gland or duct theory has enjoyed the greatest current popularity. This is based chiefly upon the fact that they usually arise from major bronchi and rarely from the smaller ramifications which do not contain mucous glands. However, transitions from mucous glands to carcinomas have not been reported. The fact that the overlying mucosa is usually intact and separated from the tumor by a band of connective tissue has also been mitigated against a mucosal origin. Many authors seem to neglect the fact that bronchial epithelium, as well as bronchial glands, contains mucous cells.¹⁵ This case showed definite transitions from normal mucosa to tumor. Our findings do not disprove the bronchial gland or duct origin for

most adenomas they do suggest that similar tumors may arise from bronchial epithelium as well. The findings of squamous metaplasia in ducts in the region of the neoplasm has been observed by others^{3, 6}

The histologic changes in this case would seem to provide additional evidence that the term bronchial adenoma is a misnomer. Nevertheless, similar situations are encountered in adenomas of other organs, such as the thyroid or kidney, where a benign appearing neoplasm may develop into frank carcinoma or may metastasize without any change in its benign histologic appearance. Fatalities due to bronchial adenomas have been due almost exclusively to secondary pulmonary changes, even in those unusual cases with metastases, unless there has been transformation into frank carcinoma. Thus it would seem desirable to retain the term adenoma until a more satisfactory one is found, provided their malignant possibilities are borne in mind. To label them as carcinomas would confuse them with typical bronchogenic carcinomas, which are so dissimilar both morphologically and clinically.

SUMMARY AND CONCLUSIONS

1 A case of peripherally located bronchial adenoma with histologic evidence of transformation into adenocarcinoma six years following its detection has been described.

2 The tumor appeared to arise from multiple foci in the bronchial mucosa.

3 The advisability of retaining the term bronchial adenoma has been briefly discussed.

4 Proved adenocarcinomatous change in a bronchial adenoma further emphasizes the necessity of treating adenomas by pulmonary resection.

5 Finally, cases such as this again raise the question of the acceptability of the policy of "watching" round lesions, thought to be tuberculous in nature.

Acknowledgment The photomicrographs were taken by Mr. Mort Russin.

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INDUCED PULMONARY TUMORS

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It is generally agreed that surgical treatment of carcinoma of the lung is limited by the advanced stage of the disease when it is first seen. More information concerning the etiology of this neoplasm is certainly needed to extend the benefits of surgical therapy and perhaps provide new types of treatment. A study of the causes of experimental pulmonary tumors in animals is one way of approaching the problem and such an investigation has been made in order to determine whether different agents known to increase the number of these tumors act in a similar manner.

The most useful animal for experimentation on tumors is the mouse and numerous strains which have been inbred for many generations over a period of years are available for study. The A strain is genetically homogeneous and spontaneously develops pulmonary adenomas which are markedly increased in numbers when carcinogenic hydrocarbons^{1, 2, 3} or nitrogen mustard⁴ or urethane¹⁰ are administered. Since many other strains are not thus susceptible, and the tumors appear regularly from one generation to the next it is believed that genes for susceptibility to these tumors are present on the chromosomes of this strain. Certain less susceptible strains of mice have visible anomalies each due to a specific gene on the chromosomes. The W strain carries three of these mutant genes.

After appropriate crosses between the A and W strains the physical appearance and the number and distribution of tumors in the offspring afford sufficient information to tell whether the genes for susceptibility to tumors are linked to genes for the visible anomalies. By comparing results when different chemicals are used to induce the tumors it should then be possible to tell whether the tumorigenic effects of different chemicals are mediated through a similar pathway. Results of such linkage studies on mice with pulmonary adenomas induced with urethane are presented in this report.

METHODS*

Three mutant genes having visible manifestations in the W strain are *haker 2*, *sh 2*, and *folded tail 2* and are designated as *sh 2*, *wa 2*, and *f* respectively. All are recessive and must be present on both chromosomes of the pair before a mouse will show the characteristic. Deafness, choreic motions of the head, and occasional whirling movements are characteristic of *haker 2*. The coat is curled in the presence of *waved 2* and the manifestations of *folded tail 2* are siderostic anemia, flexion of the tail, and a white spot on the abdomen. *sh 2* and *folded tail 2* and *shaker 2* to be located on the same chromosome. The A strain carries the dominant normal genes *sh 2*, *wa 2*, and *f* at these loci.

* From the Department of Surgery, Louisiana State University School of Medicine.
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Mice of the *A* strain were mated to the *W* strain and the offspring, having one of the genes under discussion from each parent, therefore had the genetic constitution *Sh 2/sh 2*, *Wa 2/wa 2*, *F/f*. Females of this type were then backcrossed to the *W* strain, and among the resulting offspring it was possible to compare *Sh 2/sh 2* to *sh 2/sh 2*, *Wa 2/wa 2* to *wa 2/wa 2*, and *F/f* to *f/f* individuals. Mice of these genotypes have the following physical appearance, respectively *Sh 2*, *sh 2*, *Wa 2*, *wa 2*, *F*, and *f*. This designation will be used in the description hereafter rather than giving the entire genotype.

The animals were injected with urethane at weekly intervals beginning when the mice were ten weeks of age, and continuing for fourteen weeks. One milliliter of recrystallized urethane in 10 per cent aqueous solution was injected intraperitoneally in weekly dosages of 1 ml per 100 Gm. of body weight. Weights were recorded at weekly intervals throughout the



Fig 1—Pulmonary adenoma in the *A* strain

study. The mice were autopsied four weeks after the completion of treatment, and the number of pulmonary tumors counted under the dissecting microscope. They were counted again after fixation as a check on the accuracy of the tabulation. Sections were prepared, and histologic study made of the lungs from each mouse. A control group of *A* mice were also given the urethane treatment described and were studied in a similar manner.

RESULTS

Urethane proved to be somewhat toxic in the dosage used, but 91 per cent of the animals survived the treatment. All of those completing 14 injections and surviving four additional weeks were found to have one or more adenomas.

This was confirmed by examination of microscopic sections. The gross appearance of the tumors is illustrated by Fig. 1, and a microscopic section of one of the tumors protruding into the bronchus appears in Fig. 2. In some instances the tumors were exceedingly numerous and involved all lobes of the lung, with a few tumors in some specimens becoming confluent. The number of tumors was checked by weighing the lungs from each mouse. As might be expected, more tumors usually occurred in the lungs weighing the most. There was an average of 34.6 pulmonary adenomas per mouse, with 35.8 in females and 31.2 in males.



Fig. 2.—Microscopic section of the tumor shown in Fig. 1. Note protrusion of tumor into bronchus.

The mean weight of the lungs was 0.217 Gm. The mean number of tumors in mice with lungs of greater than average weight was 45.1, whereas the mean number of adenomas was only 26.0 in those mice with lungs weighing less than the average value. In a control group of 53 mice of the A strain, the average number of adenomas per mouse was 83.7, with an average of 85.0 tumors in 30 females and 81.2 tumors in 23 males.

Data for those animals surviving twenty-eight weeks are arranged in Table I so that the tumor count in those mice showing visible manifestations of marker genes may be compared to those without. In the backcross, *Sh* 2 mice had an average of 36.1 tumors, whereas *sh* 2 animals had only 25.1. This is a statistically significant difference ($P = 0.0001$). Likewise, the average number of tumors was 35.4 for *Wa* 2 animals, which was significantly higher than the average, 25.6, for the control group ($P = 0.0007$). On the other hand, the mean number of tumors for *F* (nonfixed tail) mice, 31.1, is not significantly different from the

WEIGHT CURVES OF BACKCROSS MICE TREATED WITH URETHANE

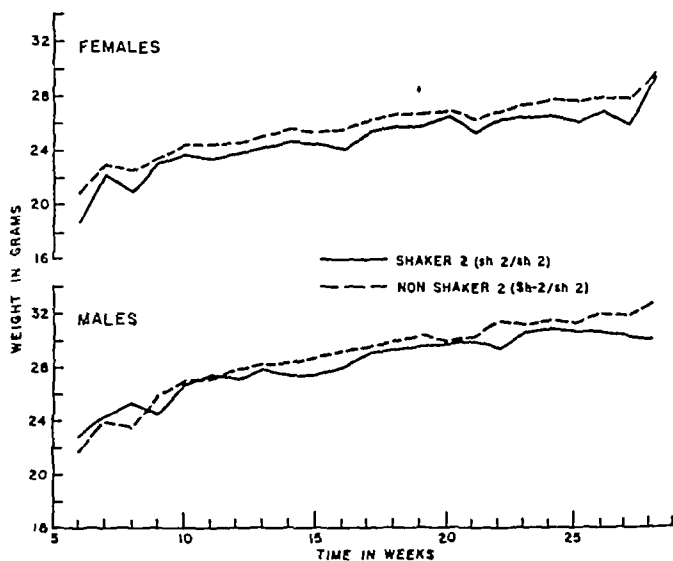


Fig 3—Comparison of weight curves of mice with and without shaker 2

WEIGHT CURVES OF BACKCROSS MICE TREATED WITH URETHANE

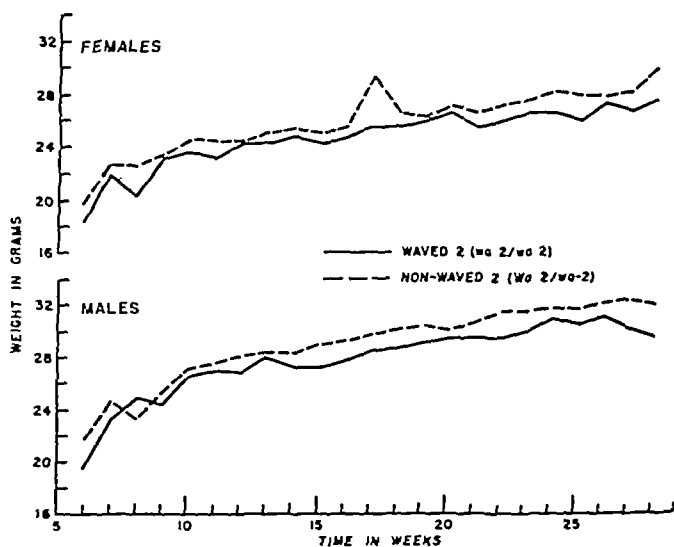


Fig 4—Comparison of weight curves of mice with and without waved 2

mean for *f* (flexed-tail) individuals, 32.5 ($P = 0.19$). Similar differences are found between the medians of the various groups and the means for the right (upper, middle, and lower), right median, and left lobes separately.

Curves for the average body weight of the various groups are shown in Figs 3 to 6. Although the weights are consistently lower in those mice having *coat-2* coats, *shaker-2* behavior, and a belly spot, the difference is small compared to the range of weights. Females with flexed tails have mean weights very similar to those without, but males with flexed tails have average weights which are less than those with straight tails (Fig. 3).

WEIGHT CURVES OF BACKCROSS MICE TREATED WITH URETHANE

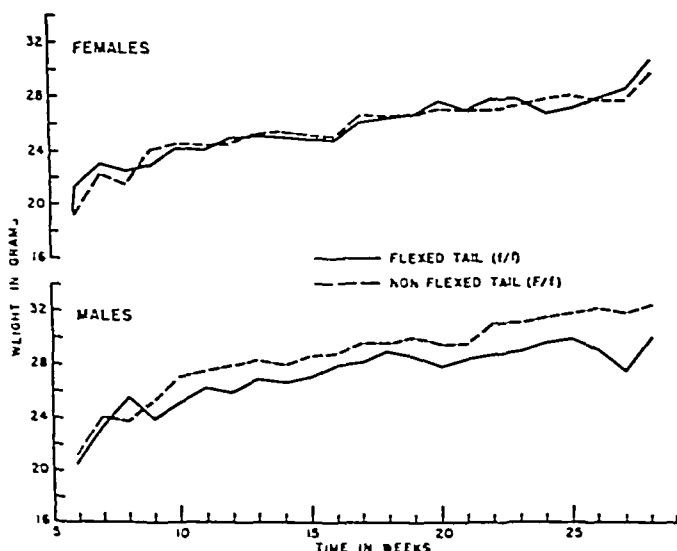


Fig. 3.—Comparison of weight curves of mice with and without flexed tail

DISCUSSION

There is good evidence that greater numbers of pulmonary tumors appear after urethane treatment when the *Sh-2* and *Wa-2* genes are expressed than when *sh-2* and *wa-2* are present on both chromosomes of the pair (Table I). It is possible that the results for the *Sh-2* and *Wa-2* loci are due to double or secondary effects of each gene, the presence of a susceptibility gene adjacent to a respective marker gene, or the presence of a susceptibility gene (or genes) somewhere on the chromosome bearing the two markers. Even if only one susceptibility gene were present on the chromosome, the data suggest it to be at a different locus than that associated with *Sh-2* and described by Heston in his experiments with methylcholanthrene. Cowan² did not determine linkage, but suggested susceptibility to one dominant gene when urethane was used for induction of tumors in hybrids of the *A* and *C* stocks.

Mean body weight is slightly and somewhat consistently lower in groups of mice with the *sh-2* and *wa-2* characteristics, but the difference is not sufficiently large or the corresponding range in weights sufficiently narrow to be considered decisive (Figs 3 and 4). Also there is a difference between the weights of males and females, and yet the incidence of tumors, if different at all, is lower in males than females. Therefore there is no conclusive evidence that the greater number of pulmonary tumors in *Sh-2* and *Wa-2* mice is secondarily related to the effect of the genes on body weight.

WEIGHT CURVES OF BACKCROSS MICE TREATED WITH URETHANE

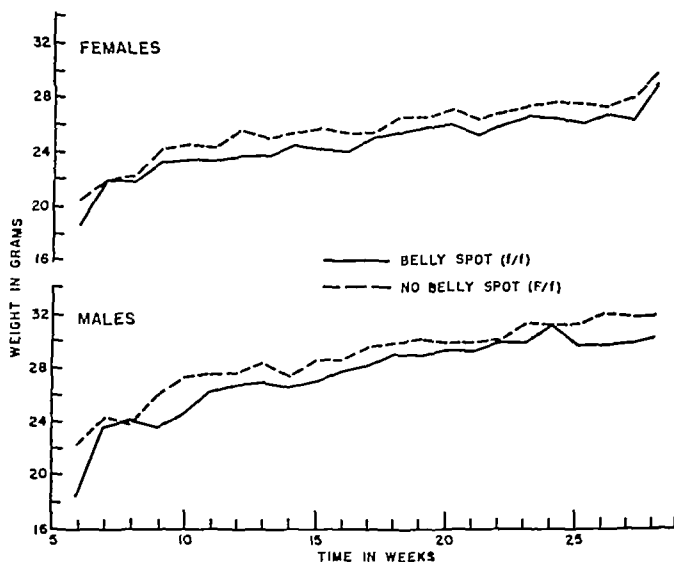


Fig 6—Comparison of weight curves of mice with and without belly spot.

No difference is found in the average number of tumors when mice with flexed tails are compared to those without. However, it is interesting that a difference in tumor count becomes apparent as soon as the presence of a belly spot is used as the criterion of the presence of the *f* gene. The decreased incidence of tumors in animals with a belly spot apparently is not an example of true linkage to the *f* gene, but is an association with one of its manifestations. Again the weights encountered do not entirely correspond to expectations if the effect of the gene on tumor incidence is mediated through its effect on body weight. Differences in body weight are small (Figs 5 and 6) and there is a difference in the weights of males but none in the weights of females when a comparison of the weights of mice with flexed tail and nonflexed tail is made.

Heston⁸ reports linkage to *Sh-2* and *F* (nonflexed tail) but not to *Wa-2* when 20 methyleholanthrene was used to induce pulmonary tumors. In contrast to these findings, the data in this report indicate linkage to *Sh-2* and *Wa-2* and

TABLE I. LINKAGE OF SUSCEPTIBILITY TO PULMONARY ADENOMAS INDUCED WITH URETHANE

STRAIN OR GENERALIZATION	MARKER GENES	NO OF MICE INJECTED	NUMBER OF TUMORS		MEAN TUMORS PER LOBE			
			MEDIAN	MEAN	P	RIGHT†	MEDIAN	LEFT
A		53	84	83.7 ± 2.75*				
BC ₁	Sh 2	127	32	36.1 ± 1.92	0.0001	21.6	3.3	11.2
BC	sh 2	97	21	25.1 ± 1.59		14.6	2.3	8.1
BC	Wa 2	132	31	35.4 ± 2.09	0.0007	21.1	3.3	11.2
BC	wa 2	92	22	25.6 ± 2.38		15.1	2.2	7.8
BC	F	179	27	31.1 ± 1.58	0.19	18.6	2.9	9.7
BC	f (Flexed tail)	45	28	32.5 ± 3.38		18.8	2.7	9.8
BC	f	148	30	34.4 ± 1.77	0.003	20.1	3.1	10.9
BC	f (Belly spot)	76	23	25.5 ± 3.58		15.8	2.2	7.6

*Standard error of the mean.

†Upper middle and lower

‡Backcross.

none to *F* (nonflexed tail) when urethane was used. Therefore, the pathway by which the two chemicals act must not be entirely the same, since genes for susceptibility to the chemicals do not show the same linkages.

SUMMARY

1 Resistance to pulmonary adenomas induced with urethane is linked to the *shaker-2* and *waved-2* genes on one chromosome in the mouse but not to the flexed tail manifestation of an additional gene on another chromosome.

2 Since linkage of susceptibility genes and certain marker genes is not the same for tumors induced by urethane and 20-methylcholanthrene, it is likely that these two chemicals do not act in an entirely similar manner in producing the same type of tumor.

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Announcement

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The 33rd annual meeting of the American Association for Thoracic Surgery will be held March 27, 28, and 30, 1953, in San Francisco, Calif. Headquarters will be at the Hotel Fairmont. Suitable accommodations also have been reserved at near by hotels, the Mark Hopkins, the Huntington and the Sir Francis Drake

Requests for Hotel Reservations

These should be mailed to Dr. H. Brodie Stephens, Chairman of Hotel Committee, American Association for Thoracic Surgery, Room 200, 61 Grove St., San Francisco 2, Calif. The following information must be included: (1) two choices of hotels, (2) type of accommodation desired—single, double, twin, room or suite, (3) name of other room occupants, if any, whether wife or another doctor, (4) date and approximate hour of arrival, (5) date of departure, (6) return address

Abstracts for Papers

Abstracts for the presentation of papers at the 1953 meeting must be received on or before Dec. 1, 1952, otherwise they will not be considered by the Program Committee. *Five, repeat, five copies of each abstract should be sent to the Secretary of the Association, Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif.* They should not be sent to the Editor.

The abstracts should contain from 200 to 250 words, and should accurately reflect the contents of the completed paper

Applications for Membership

Applications for Associate Membership in the American Association for Thoracic Surgery must be received by the Membership Committee not later than December 1, 1952, to be considered for action at the meeting in San Francisco in March, 1953. Application received on a date beyond that deadline must be deferred for consideration until 1954.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the sponsoring statement on the application form, the sponsors must submit a separate letter concerning the applicant direct to the Chairman of the Membership Committee. These regulations are at the direction of the Council and were made effective at the meeting in Dallas in 1952.

Completed application forms should be sent to the Chairman of the Membership Committee, Edward M. Kent, M.D., 1406 Clark Building, Pittsburgh 22, Pa.

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Original Communications

THE CARE OF THORACIC AND THORACOABDOMINAL WOUNDS IN THE COMBAT ZONE IN KOREA

MAJOR EDWARD LERNEST ROCKEY * MEDICAL CORPS ARMY OF THE UNITED STATES

THIS study is based on 163 thoracic and thoracoabdominal injuries handled at the Mobile Army Surgical Hospital 8076 Army Unit in Korea between July 17 and Dec. 17, 1951.

The missile wounds were penetrating in 126 cases (77.3 per cent), perforating in 32 cases (19.6 per cent) and the remaining 5 cases (3.1 per cent) had crush injuries of the chest. One hundred and twenty-seven (77.9 per cent) of these patients had thoracic and 36 (22.1 per cent) had thoracoabdominal injuries. The wounds were sucking at the time of admission or during the process of debridement in 43 cases (26.4 per cent). The pathology was unilateral in 152 (93.3 per cent) and bilateral in 11 (6.7 per cent) of the patients. Of the 152 unilateral injuries 86 (57 per cent) were located on the right and 66 (43 per cent) on the left side.

PREOPERATIVE CARE

At the time of admission the wounds were carefully surveyed. Physical examination, posteroanterior and lateral x-ray views of the chest were utilized in the evaluation of the patient. Blood pressure, pulse, and the rate of respiration were checked at frequent intervals. Blood transfusion was started without delay unless there was no definite evidence of appreciable blood loss. All the patients were given antibiotics, namely, one million units of crystalline penicillin 11 Gm. of streptomycin at the time of admission followed by 600,000 units of penicillin every six hours and 1 Gm. of streptomycin twice a day during the stay in the hospital. The patients were kept in a position most comfort-

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able to them which frequently was Fowler or semi-Fowler. Those in shock were kept flat on their backs or in the Trendelenburg position. Oxygen was administered as indicated through nasal catheters.

CLINICAL MATERIAL

Table I is a summary of the clinical material and indicates the type of pathology represented, and the form of therapy applied with the results.

TABLE I A SUMMARY OF THE CLINICAL MATERIAL

FINDINGS	PENETRATING WOUNDS	PERFORATING WOUNDS	CRUSH INJURIES	EVACUATED	DIED	TOTAL
Retained metallic foreign bodies without hematoma, hemothorax or pneumothorax	15			15		15
Hematoma	9	3		12		12
Hemothorax, hemopneumothorax or pneumothorax treated by repeated aspirations	38	7		45		45
Crush injuries			5	5		5
Sucking wounds of the chest treated by tube aspiration and closure of the wound	13	9		22		22
Conditions requiring thoracotomy or its modifications	11	6		14	3	17
Thoracoabdominal injuries submitted to laparotomy and suction through the diaphragm	17	2		16	3	19
Suspected thoracoabdominal injuries with negative laparotomy findings	10	1		8	3	11
Conditions requiring thoracoabdominal exploration	9	4		10	3	13
Independent chest and independent intraabdominal injuries	4			4		4
Totals	126	32	5	151	12	161

I THORACIC INJURIES

Some of the penetrating missile wounds of the chest required no other treatment than débridement of the entrance wound or wounds. They were the ones with the metallic foreign body retained in the lung without evidence of hematoma, hemothorax, or hemopneumothorax on x-ray pictures. Clinically these patients were asymptomatic. They were kept under observation for two or three days prior to their evacuation. There were fifteen such cases in this series.

Twelve of the patients had no other symptoms than hemoptysis and at times pain on the side of the injury. X-ray pictures of the chest showed one or more well-defined areas of density within the parenchyma without evidence of hemothorax or hemopneumothorax. The wounds were débrided, the tracheo-

bronchial tree was kept free by encouraging coughing and expectoration, and, if needed, by bronchial catheterization or bronchoscopic suction. The hematoma of the lung in these cases cleared or regressed to a great extent prior to evacuation within three to seven days.

Forty-five of the penetrating and perforating missile wounds which led to hemothorax or hemopneumothorax were submitted only to debridement of the wounds and repeated aspiration of air and/or blood from the pleural space. The aspirations were performed as soon as the evaluation of the case was completed. As much as 1,000 c.c. of blood was aspirated at one time in many of the cases, and, if x-ray pictures showed evidence of reaccumulation of blood, the aspiration was repeated daily or, if necessary, even on the same day. Prior to the removal of the aspirating needle $\frac{1}{2}$ Gm. of streptomycin and 400,000 units of penicillin were instilled into the pleural space following each aspiration. The patients with hemopneumothorax or pneumothorax were submitted also to deflation of the pleural space. This was accomplished by the application of suction (by an electric suction machine) to the aspirating needle. The suction at times was continued as long as fifteen minutes. These patients showed evidence of good lung re-expansion. The clouding caused by the hemothorax cleared completely, or to a great extent, prior to evacuation within two to eight days with the exception of two patients. In these two cases the need for surgical evacuation of clotted, or perhaps organized blood from the pleural space was obvious.

Five patients were seen with crush injuries represented by multiple rib fractures, hemothorax, and hematoma of the lung. Intercostal nerve block with 1 per cent procaine, repeated aspiration of the hemothorax, and keeping the air passages clear by repeated catheter aspirations stabilized these patients. They all were evacuated in good condition and their lungs expanded within three to eight days following admission.

The open or sucking wounds of the chest or wounds suspected of being such, with hemothorax or hemopneumothorax were handled under general endotracheal anesthesia. The wounds were debrided, a thoracotomy tube was inserted through the debrided wound, and the pleural space was aspirated. The wound was closed up to skin level while the lung was kept inflated and the temporarily inserted thoracotomy tube was removed simultaneously. As indicated by subsequent x-ray findings, some of these patients needed further daily needle aspiration. Those sucking wounds which presented evidence of depressed rib fractures required resection of the irregular rib ends. In these cases the pleural space was irrigated with normal saline solution prior to the closure of the chest wall defect by approximation of the soft tissue layers outside of the parietal pleura. Also, a thoracotomy tube was inserted into the pleural space in a dependent position for continuous underwater drainage. Twenty-two of forty-three sucking wounds of the chest were treated only by this method. All fully re-expanded lungs with only evidence of clearing parenchymal clouds at the time of their evacuation. The rest of the sucking wounds fell into the group of thoracoabdominal injuries.

Conditions requiring open thoracotomy or modifications of it at a Mobile Army Surgical Hospital level are few (1) active intrapleural bleeding from injured lung with or without internal mammary or intercostal vessel injury, (2) injury of mediastinal structures, (3) presence of large intrapleural metallic foreign body, if it is easily accessible from the site of the entrance wound, (4) wounds of the large bronchi and intrathoracic part of the trachea, (5) wounds of the heart

Contrary to the writings of Mackler,¹ Tuckwell,² Woodruff,³ Blackburn,⁴ and others, perforation or anatomic likelihood of perforation of the diaphragm was not found to be an indication for open thoracotomy

A total of seventeen patients were submitted to open thoracotomies. Three of them died in the operating room, one, of perforation of the aorta, two, of heart wounds. The cardiorespiratory function of the two patients with heart wounds ceased before the incisions were made and the thoracotomies were performed primarily for purposes of resuscitation. The remaining fourteen patients were evacuated in good condition with the lung well expanded and aerated, showing evidence of parenchymal clouding only at the site of the repaired lung wounds. In four of them the chest was entered through extensions of the chest wall wound. The remaining thirteen patients were operated on through elective incisions. In four of the patients a posterolateral approach was used, and in nine of them the chest was entered through anterior incisions (Diagram 1, A, B, C, and D). In this procedure the pleural cavity was entered through the third, fourth, or fifth anterior intercostal space, and correspondingly the third and fourth, or fourth and fifth, or fifth and sixth cartilages were divided. The skin incisions were placed over the intercostal space which was to be utilized as an entrance to the pleural cavity, and were extended from a point about one inch medial to the costochondral junction to the midaxillary line. If the plan was to divide more than two cartilages the incision was extended downward at a right angle to its medial end (Diagram 1, B, C, and D). A meticulous closure of the wound in layers assured good healing. The lung lacerations were carefully débrided and the bleeding points controlled by suture ligatures of 00 or 000 silk. The bronchial openings were closed with 00 silk mattress sutures. The débrided lung surfaces were approximated in layers with 00 chromic catgut sutures. The perforating wounds of the lung were handled in a similar fashion except that in the tunnel of the wound strips of Gelfoam were inserted. Only nonviable subsegments or segments of the lung were resected. In none of the cases was lobectomy or pneumonectomy performed. Prior to the closure of the chest wall the pleural space was carefully irrigated with normal saline solution, the lung was fully reexpanded, and two thoracotomy tubes were inserted into the pleural space for underwater drainage.

II THORACOABDOMINAL INJURIES

As stated previously perforation of the diaphragm is not an indication for open thoracotomy. These thoracoabdominal injuries were handled by laparotomy preceded by closure of the sucking wound of the chest, if such existed. The diaphragmatic perforation was closed from the abdominal side

while the contents of the pleural space were aspirated by a thoracotomy tube which was inserted into the pleural space through the tear of the diaphragm. The tube, to which constant suction was applied, was removed while the last suture of the diaphragm was tied and the lung kept inflated, simultaneously. In cases where a hollow viscus of the abdomen was perforated, the pleural space was carefully irrigated with saline solution prior to the closure of the diaphragmatic tear, frequently a thoracotomy tube was inserted into the pleural space usually after the completion of the laparotomy.

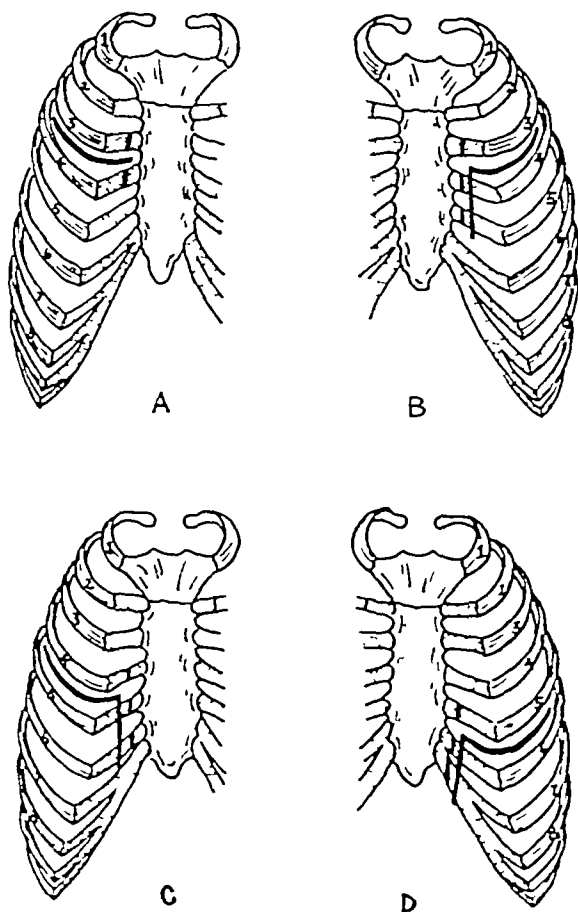


Diagram 1—Varieties of thoracotomy incisions

Nineteen of our patients were handled in this fashion. Sixteen of them were discharged in good condition with the lung well expanded within ten days, following their operation. Three of the patients died: one in the operating room, one two days following the operation, of irreversible shock and renal failure, and one, six days following the operation, of adhesive ileus and pneumonia.

There were eleven suspected thoracoabdominal injuries which were subjected to laparotomy with negative intra-abdominal findings. If the abdominal findings were unilateral, intercostal nerve block served as a great help in differentiating referred thoracic signs and symptoms from true abdominal pathology. Eight of these patients were evacuated in good condition with the injured lung well expanded. Three of them with bilateral chest wounds died. Each of these patients had sucking wounds which were taken care of prior to laparotomy. All three of them died of respiratory failure caused by extensive bilateral intrapulmonary hemorrhages. In each case x-ray examination showed well defined areas of clouding of both lung fields. The patients expectorated considerable amounts of blood. Two of them died on the first, and one on the second, postoperative day. Two of these patients were autopsied confirming the clinical and x-ray findings of extensive bilateral pulmonary hepatization.

Conditions requiring thoracoabdominal exploration or modifications of it are few. These are upper abdominal and lower thoracic injuries not accessible through laparotomy wounds alone such as

(1) some of the liver wounds, (2) few of the otherwise not accessible diaphragmatic tears, (3) injuries of the lower end of the esophagus and cardiac end of the stomach.

These conditions, of course, are in addition to the cases with indications for open thoracotomy which also have evidence of intra-abdominal injury.

Thirteen patients were in this group of which only eight were true thoracoabdominal explorations. Three of them were submitted to thoracotomy followed by laparotomy. Two of them had laparotomies and débridement of chest wall defect leading to adequate exposure to correct the intrathoracic part of the pathology. Ten of the patients did well and were evacuated with well expanded lungs, showing x-ray evidence of hematoma only at the site of the repaired lung injury. Three of them died: two in the operating room of cardiac standstill, and one of adynamic ileus on the seventh postoperative day.

The operative approaches to abdominothoracic exploration are indicated in Diagram 2, A, B, C, and D. A midline, or occasionally a paramedian laparotomy incision was always used. After the abdominal cavity was opened and the indication to enter the chest was established, the operative incision was extended upward through the seventh, sixth, and fifth cartilages, and at times even the fourth. If needed, a lateral extension was made to the midaxillary line within the intercostal space below the highest divided cartilage (most commonly the fifth). In the majority of the cases this lateral extension of the incision was not necessary (Diagram 2, D). At times, the diaphragm was divided up to the tear or perforation in order to increase the visibility posteriorly. In cases of urgency in the thoracic part of the injury, as in cases of active bleeding, the operation can be started as an anterior thoracotomy and later the incision extended downward.

Independent chest injuries with independent abdominal injuries resulting from multiple penetrating shell fragment wounds were handled as indicated. The principle of early reestablishment of normal cardiorespiratory physiology in so far as possible was kept in mind. The chest injuries were first treated

thus increasing the chances that these patients would survive a subsequent immediate laparotomy. Four patients were handled in this manner. All of them were evacuated in good condition with the injured lung fully expanded.

POSTOPERATIVE CARE

In the postoperative period the antibiotic therapy was maintained until the evacuation of the patients, which was approximately ten days. The thoracotomy tubes were frequently suctioned, in addition to the continuous underwater drainage. Breathing exercises and coughing were encouraged. Bronchial catheterization and bronchoscopic suction were applied without hesitation. Needle aspirations were performed if the x-ray findings suggested reaccumulation of intrapleural fluid.

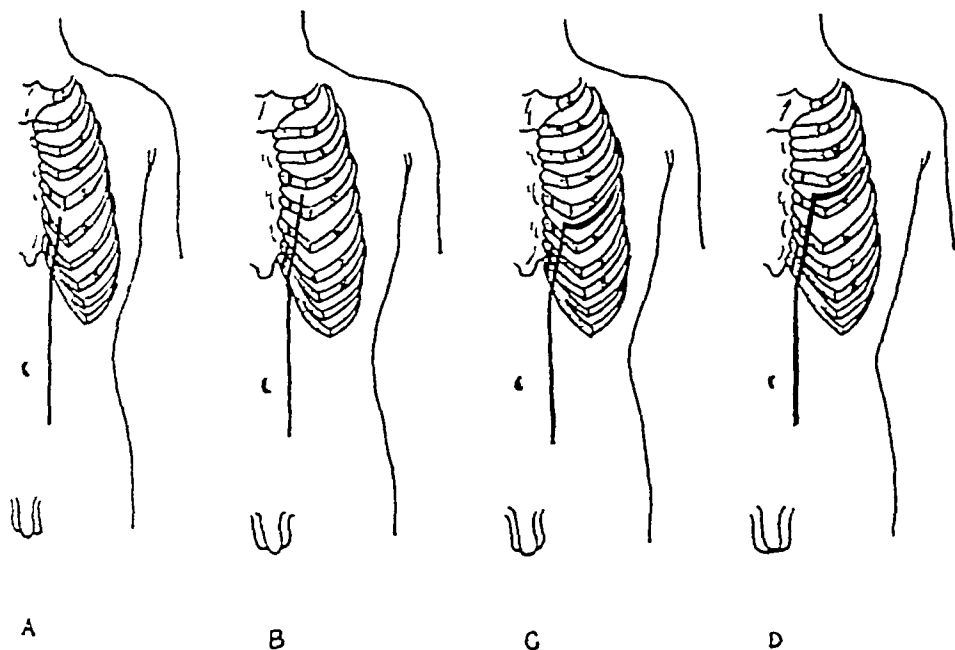


Diagram 2—Varieties of abdominothoracic incisions

ILLUSTRATIVE CASES

CASE 1.—A Korean American corporal sustained multiple shell fragment wounds of the chest and abdomen. One metallic foreign body was retained in the anterior chest wall in the right hilar region. Posteroanterior and lateral x-ray views (Figs. 1 and 2) failed to reveal hemothorax, pneumothorax, or evidence of interpleural artery bleed. X-ray pictures in two days prior to evacuation showed no change in the appearance of the lungs. This patient required no active therapy other than treatment of the wounds.

This is representative case of penetrating missile wounds without deleterious consequences.

CASE 2—E T, an American corporal, sustained crush injury to the right chest in a truck accident. He had moderate subcutaneous emphysema. X ray pictures showed fracture of the first to seventh ribs inclusive on the right side and evidence of minimal hemothorax. Intercostal nerve block with 1 per cent procaine was done from the first to the ninth inter

Fig 1



Fig 2



Fig 3



Fig 4

Fig 1—A metallic foreign body is located at the level of the sixth posterior intercostal space near the hilum and one at the level of the fifth posterior rib at midline.

Fig 2—Lateral view places one metallic foreign body near the hilum and one within the anterior chest wall.

Fig 3—Oblique view of the chest shows multiple rib fractures and moderate subcutaneous emphysema.

Fig 4—Posteroanterior view shows fracture of the first to seventh ribs with the lung expanded.

tal space inclusive. Blood was aspirated from the right pleural space in five consecutive days with clearing of the previously blurred costophrenic sinus and subcutaneous emphysema to a great extent (Figs 3 and 4).

This is a representative case of crush injury which responded well to conservative measures.

Fig 5

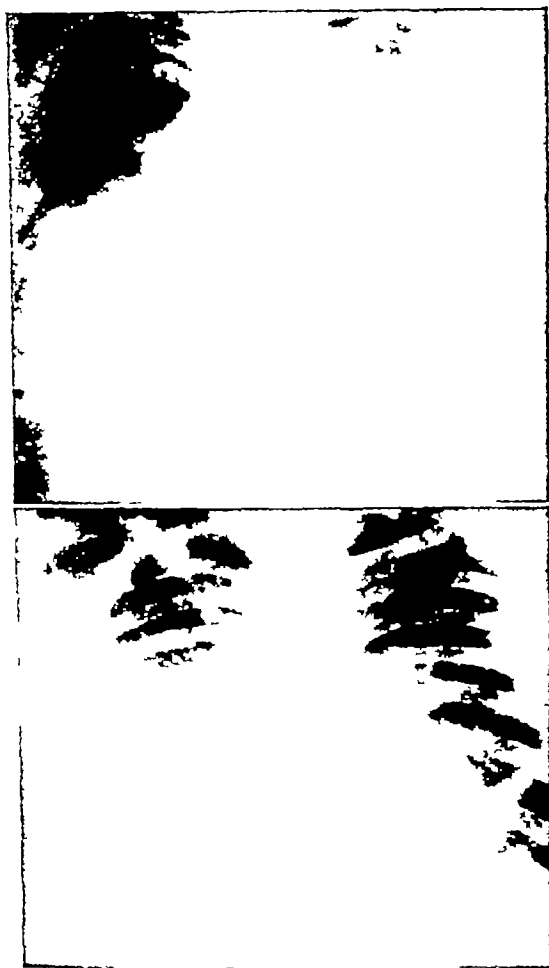


Fig 6

Fig 5.—Moderate hemothorax on the right.

Fig 6.—Considerable clearing of the right lung field with a metallic foreign body present in the lower lobe.

CASE 3. C. L. S., a 23-year-old American corporal, was admitted thirteen hours after two 1 1/2 inch fragment wounds of the back and leg. At the time of admission he complained of pain in the right chest and expectorated a moderate amount of blood. A 1 by 1 1/2 inch wound was present over the right lower rib cage wall near the paravertebral examination revealed hyperinflated breath sounds on the right side of the chest. The chest X-ray showed moderate clearing on the right with a metallic foreign body retained within the lung. Deflation of the lungs was the

Three aspirations of blood from the right pleural space led to considerable clearing of the right lung field (Fig 6) Nine days following admission, this patient was evacuated in asymptomatic condition

This is a representative case of hemothorax which responded well to repeated aspirations

CASE 4—K. Y. S., a 17 year old South Korean soldier, sustained a penetrating shell fragment wound of the left chest with the entrance wound at about the seventh intercostal space at the midaxillary line resulting in left hemothorax (Fig 7) Two successive aspirations of blood from the left pleural space led to some clearing of the lung field The patient expectorated a considerable amount of blood, and showed evidence of atelectasis of the right lower lobe two days following admission Bronchoscopic suction, encouraged coughing regime, and repeated pleural aspirations on the left side led to good clearing of both lung fields (Fig 8) He was asymptomatic at the time of his evacuation, four days following admission

This is a representative case of hemothorax and atelectasis which responded well to repeated aspirations, bronchoscopic suction, and encouraged coughing regime

CASE 5—V. F. K., an American private first class, sustained perforating bullet wounds of the left chest causing hemothorax and intrapulmonary hemorrhage within the left upper lobe region The missile entered at the fourth intercostal space and parascapular line and left the chest at about the anterior end of the first rib Aspiration revealed 1,200 cc of bright red blood, followed by 200 cc at the time of second aspiration on the same day Under general endotracheal anesthesia, the wounds were débrided and the pleural space was aspirated by a thoracotomy tube through the débrided wounds which were sucking at this stage of the operation As the tube was removed, the wounds were closed with 0 chromic catgut interrupted sutures up to skin level Three days later aspiration revealed 400 cc of dark blood The next day aspiration failed to reveal blood, and the patient was evacuated in an asymptomatic condition with the lung fully re expanded

This is a representative case of sucking perforating wounds of the chest with hemothorax and pulmonary hematoma

CASE 6—J. R. H., a 20 year old American private, sustained perforating gunshot wounds of the right chest He was admitted twelve hours after the injury, complaining of pain and dyspnea He had marked subcutaneous emphysema of the chest wall The entrance wound was located at about the level of the fourth rib in the interscapular space, and the exit wound at the right supraclavicular region and midclavicular line The latter was sucking while handled X ray findings (Fig 9) showed hemopneumothorax, comminuted fracture of the outer half of the right clavicle, and depressed fracture of the second, third, and fourth ribs Prior to surgical intervention 400 cc of blood and a large amount of air were aspirated Under endotracheal anesthesia the wounds were débrided, loose clavicle and rib particles were removed, the irregular clavicle and rib ends were resected, the pleural space was aspirated, and a thoracotomy tube was inserted into the pleural space in a dependent position The wound was closed in layers Six days following admission, he was evacuated in asymptomatic condition X ray pictures showed complete lung re expansion with only parenchymal clouding present (Fig 10)

This patient exemplifies sucking chest wounds with depressed fracture of ribs and hemopneumothorax

CASE 7—E. J. G., a young American soldier, was admitted five and one half hours after he sustained a penetrating bullet wound of the right chest At the time of admission he complained of hemoptysis and dyspnea, and had marked subcutaneous emphysema which

Fig 7



Fig 8



Fig 9

Fig 10

- Fig 7 - H n thorax on the left, atelectasis of the right lower lobe
 Fig 8 - Both lungs still appear to be normal
 Fig 9 - Distal fracture of the right clavicle, second, third and fourth ribs with
 Fig 10 - The apex of the lungs is at the level of the sixth posterior rib
 Fig 11 - The lungs are re-expanded. There is only evidence of parenchymal clouding

progressed while under observation. X-ray examination showed hemopneumothorax on the right, fracture of the sixth posterior rib, and a freely movable bullet within the pleural space (Figs 11, 12, and 13). Under general endotracheal anesthesia the entrance wound was débrided and enlarged, and the fractured rib ends were resected to sufficient length to allow the entrance of the examining hand into the pleural space. A 31 caliber Russian carbine bullet and a large amount of clotted blood were evacuated from the pleural space. This

Fig 11

Fig 12



Fig 13

Fig 14

Fig 11—Right hemopneumothorax and depressed fracture of the sixth rib

Fig 12—The bullet is at the level of the eleventh rib its point is downward.

Fig 13—Lateral view shows the bullet pointing upward

Fig 14—The right lung is fully expanded. There is evidence of some localized pleural thickening and also of some parenchymal clouding

as irrigated with saline solution, a thoracotomy tube was inserted in a dependent position and the chest wall wound was closed in layers. Six days following admission, at the time of evacuation the patient was asymptomatic and the lung was fully re-expanded (Fig. 14).

Fig. 15

Fig. 16

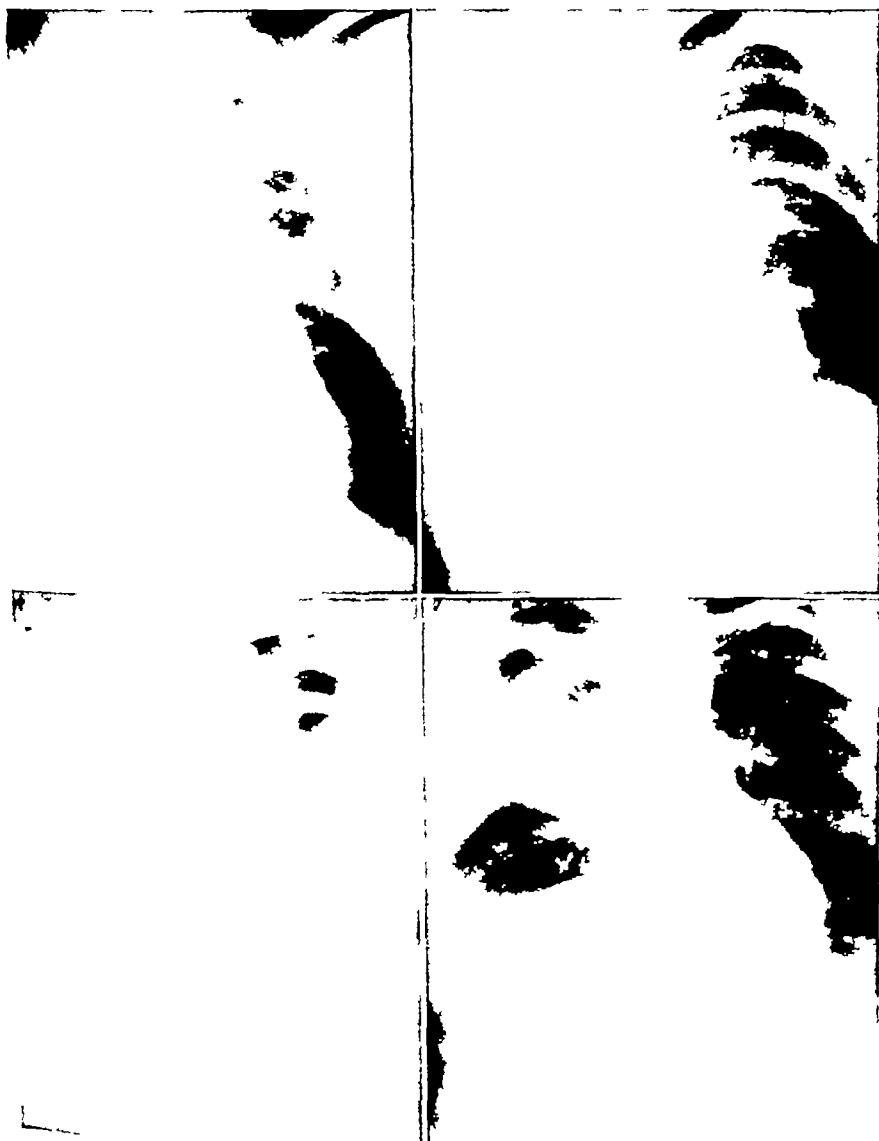


Fig. 17

Fig. 18

Myocardial infarction on the right. Fracture of the third and fourth ribs.
The right lung is expanded. There is evidence of clapping at the site of the re-
fracture. All no. of fluid is at the right of the fourth posterior in-
tercostal space on the right.
The right lung is fully re-expanded and shows evidence of paralytic atelectasis of the right lung.

This is a case with intrapleural metallic foreign body The patient was submitted to a modified thoracotomy

CASE 8—H L. S., a 21 year old American sergeant, was wounded by a hand grenade which exploded in his right hand He was admitted 6 and one-half hours following the injury in shock, acute respiratory distress, a bleeding and sucking wound of the right anterior chest, avulsion of the right hand, and shell fragment wound of the jaw The sucking, bleeding wound measured about $2\frac{1}{2}$ by $1\frac{1}{2}$ inches and was located at the anterior end of the first rib X ray view (Fig 15) showed massive hemothorax on the right, and fracture of the third anterior rib at the midaxillary line The patient received 1,500 cc of blood before he was taken to the operating room. Under general endotracheal anesthesia an open thoracotomy was performed with anterior approach A "Y" shaped fracture of the liverlike upper lobe was found almost completely dividing the lower half of the anterior and posterior segments of the upper lobe in an anteroposterior and lateral direction The torn lung surfaces were bleeding profusely One metallic foreign body was removed from the lung The bleeding points were controlled with 000 silk ligatures The débrided lung surfaces were approximated with 00 chromic catgut mattress sutures with the incorporation of eight Gulfoam strips The visceral pleura in the area of the tear was also approximated The pleural space was evacuated and irrigated, two thoracotomy tubes were inserted into the right chest cavity, and the wound was closed in layers The sucking wound of the anterior chest wall was débrided and closed, assuring full lung re-expansion at the conclusion of the procedure Also a circular amputation of the right midforearm was performed At the time of the patient's evacuation eight days following admission, the lung was re-expanded with the exception of a small anterior upper lung field pocket of fluid (Fig 16) from which 40 cc serous fluid was aspirated. According to a follow up report, two months later, both lung fields appeared to be normal on x ray examination

In this case, active intrapleural bleeding was the indication for formal thoracotomy

CASE 9—W R., a 23 year old American private, wounded himself accidentally while cleaning his rifle He sustained perforating bullet wounds of the right chest The patient was admitted in shock two hours after the injury The entrance wound was at the costochondral junction of the third and fourth anterior ribs, and the exit wound just outside of the scapula, at about the level of the fourth posterior rib Roentgenogram showed massive hemothorax on the right (Fig 17) Aspiration revealed 820 cc of blood, and 800 cc next day, with no appreciable change in the appearance of the right lung field He received 2,500 cc of blood during the period of observation His general condition improved somewhat though the blood pressure remained 90/70 It was felt that we were dealing with active intrapleural bleeding An open thoracotomy was performed through a posterolateral approach The lower part of the anterior and posterior segment of the upper lobe was traversed by the bullet tract from which oozing of bright red blood was noted In the middle of the bullet tract a cartilage about 1 by 1 cm in diameter was found in addition to dirt Near the lower margin of the entrance wound the internal mammary artery was torn, and found to be spurting into the pleural space The bleeding internal mammary artery was ligated The lung wound was débrided, irrigated with normal saline solution, and repaired with 00 chromic catgut interrupted mattress sutures Two thoracotomy tubes were inserted into the pleural space, and the operative wound was closed in layers The entrance and exit wounds were débrided and closed up to skin level The patient made an uneventful recovery, and ten days following surgery, he was evacuated in an asymptomatic condition. The wound was healed and the lung was fully re extended, showing only parenchymal clouding at the site of the débrided and sutured lung wound (Fig 18)

Intrapleural active bleeding was the indication for open thoracotomy in this case

CASE 10—A D, a 20 year old American sergeant, had sustained perforating missile wounds twelve hours before admittance. The bullet entered at the left side of the neck and entered the mediastinum, right chest, and right upper arm. He was in marked respiratory distress and had extensive subcutaneous emphysema of the neck above the sternum and right side of the chest wall. He appeared to be moribund. The entrance wound (1½ cm. in diameter) was located at about the mid part and just posterior to the sternocleidomastoideus muscle on the left side. The exit wound, about 2 by 1 inches in diameter, was at the posterior surface of the right upper arm. An x-ray view showed hemopneumothorax and fracture of the third anterior rib on the right (Fig. 19). Open thoracotomy was performed with anterior approach through the third intercostal space, followed by exploration of the left side of the chest and tracheotomy. The bullet entered the left side of the neck, perforated the left lobe of the thyroid gland, tore an area nearly 5 mm. in diameter on the anterior wall of the cervical trachea, perforated the upper mediastinum and the upper lobe of the right lung, fractured the third anterior rib, changed its direction through the axilla, and perforated the right upper arm, leaving it, over its posterior surface. At the time the mediastinal pleura was opened a considerable amount of air was escaping from the upper mediastinum. Its fracture were carefully inspected and two large Penrose drains were placed into the mediastinum the ends of which later were brought out through the neck. The perforating wounds of the upper lobe were debrided, the 2 inch long tract was cleansed with saline solution, the entrance and exit wounds were closed with 00 chromic catgut sutures after two strips of Gelfoam were placed into the bullet tract. The irregular ends of the fractured third rib were excised. The pleural space was irrigated, two thoracotomy tubes were placed into the pleural space and the chest wall wound was closed in layers. The left side of the neck was explored through an incision anterior to the sternocleidomastoideus muscle. The ends of the Penrose drains placed into the upper mediastinum through the chest were brought out through the neck and the. The tear of the anterior wall of the cervical trachea was debrided and through a stab wound of the anterior surface of the neck it was used for tracheotomy. The neck wound was closely approximated and the entrance and exit wounds of the bullet were debrided. The patient had considerable respiratory difficulty during the first two days after operation though the lung was fully re-expanded at this time. As soon as the air escape from the mediastinum lessened, anoxemia ceased and the color of the patient improved considerably. Six days following operation, he was discharged in excellent general condition and the lung fully re-expanded (Fig. 20) the wounds were clean and healing and the trachea functioned properly.

In this case the mediastinal perforation and emphysema represented the indication for open thoracotomy.

patient was evacuated for drainage of the right subdiaphragmatic space. The lung field remained clear, except for the compression caused by the elevation of the right diaphragm. Subsequently the subdiaphragmatic space was evacuated with uneventful postoperative course.

Fig 19

Fig 20



Fig 21

Fig 22

Fig 19 — Hemopneumothorax on the right and fracture of the third anterior rib.

Fig 20 — The right lung is fully expanded.

Fig 21 — Left hemothorax with one metallic foreign body above and two below the diaphragm.

Fig 22 — The lung is fully expanded and shows no other abnormalities than a retained metallic foreign body.

This case is an example of the type of thoracoabdominal injuries which be treated successfully by laparotomy and closure of the diaphragm from the minimal side.

Case 12—F M, a 22 year old American private first class sustained three shell fragments of the left chest each of them measuring about 2 by 2 cm in diameter. The was tender to palpation with muscle spasm present. A posterolateral chest x ray (Fig. 21) showed evidence of hemothorax on the left side with one metallic foreign located above and two metallic foreign bodies located below the diaphragm. Aspiration of the left pleural space revealed 100 cc of blood of putrid odor. Streptomycin 1/2 and 400,000 units of penicillin were instilled into the pleural space and 1 Gm. of tommen and one million units of penicillin were given intramuscularly. Under general anesthesia, laparotomy was performed through a left midrectus incision. The



Fig. 22—The large ulcer of considerable size on the anterior wall of the gastrointestinal tract.

lung was allowed to re expand fully. On the third postoperative day the thoracotomy tubes were removed. X ray examination (Fig 22) on the fifth postoperative day showed fully expanded well aerated lung fields on both sides though the general condition was poor. The abdomen was distended, bowel sounds could not be heard, and the urinary output was inadequate. The clinical picture was of adynamic ileus and uremia of which the patient died on the sixth postoperative day.

This case represents a thoracoabdominal injury with diaphragmatic tear, handled by laparotomy and closure of the diaphragm from the abdominal side.

CASE 13—C L H, a 19 year old American private first class, was admitted twenty eight and one half hours after he was wounded. He sustained perforating thoracoabdominal wounds. At the time of admission he had marked dyspnea, hemoptysis, left chest pain, and marked distention of the abdomen (Fig 23). The bowel sounds could not be heard. The entrance wound was located at the level of the fourth rib at the anterior axillary line on the left side and measured about $\frac{1}{2}$ cm in diameter, the exit wound was located at the right flank and measured $4\frac{1}{2}$ by 2 cm in diameter. X ray pictures of the chest showed no evidence of gross injury of the lung. A laparotomy was performed through a left midrectus incision. In order to expose the cardiac end of the stomach and retrocardiac space the incision was extended upward and the seventh, sixth, fifth, and fourth cartilages were divided making the procedure an abdominothoracic one. The findings were as follows: perforating wounds of the left lower lobe with an egg sized hematoma of the anteromesial basal segment of the left lobe, a perforation about $\frac{3}{4}$ cm in diameter of the left leaf of the diaphragm, near to the cardiac end of the stomach, an egg sized hematoma at about, and posterior to, the cardiac end of the stomach and lower end of the esophagus, a retroperitoneal hematoma extending from the level of the cardia to the bifurcation of the abdominal aorta, behind the left colon and duodenum, extensive laceration of the lower half of the right kidney, considerable distention of the entire intestinal tract, left hemothorax, and large hemoperitoneum. The left pleural space was evacuated, the lung wounds were cleansed with saline solution. The lower retroesophageal and retrocardiac space was explored, the hematoma was evacuated. The retroesophageal space extending to the common iliac artery, and behind the right colon and duodenum, was inspected. A right nephrectomy was performed and the entire intestinal tract surveyed. The chest wound was closed in layers. A thoracotomy tube was placed into the left pleural space in a dependent position. A Penrose drain was placed into the right renal bed and brought out through a stab wound at the right flank. The abdominal wound was closed with through and through wire sutures. The entrance and exit wounds were débrided. The patient made an uneventful recovery. X ray pictures taken two days before evacuation on the sixth postoperative day showed the lung well expanded with no gross abnormalities visible. Follow up report in one month revealed a continued uneventful course. The wound healed well, the costal arch was firm, and the patient was reported to be asymptomatic.

This case represents one of the indications for abdominothoracic exploration.

CASE 14—F H, a 27 year old American sergeant, was wounded eleven hours prior to admission. He sustained a shrapnel wound of the right flank. His general condition was fairly good. There was a wound 4 by 4 inches in diameter, at the right flank. The abdomen was tender all over with rigidity present. The urine was red. X ray view of the chest showed some clouding of the periphery of the right lung field with a large metallic foreign body retained within the upper part of the chest (Fig 24). A laparotomy was performed through a right rectus incision. A one inch laceration of the undersurface of the right lobe of the liver and contusion of the right kidney were found in addition to moderate amount of blood in the peritoneal cavity. The right kidney and ureter were explored and the intestinal tract was surveyed. One Penrose drain was placed to the liver laceration and one to the kidney capsule, both were brought out at the right flank. The operative wound was closed with through and through wire sutures. The entrance wound was débrided. An incision was

... extending about 6 inches upward, exposing the tract of the shell fragment, and the ... minutes, fractured twelfth, eleventh, and tenth ribs with a tear of the parietal pleura at ... at the costophrenic sinus. By the time this wound was cleansed, and loose bony particles

Fig 24



Fig 25.

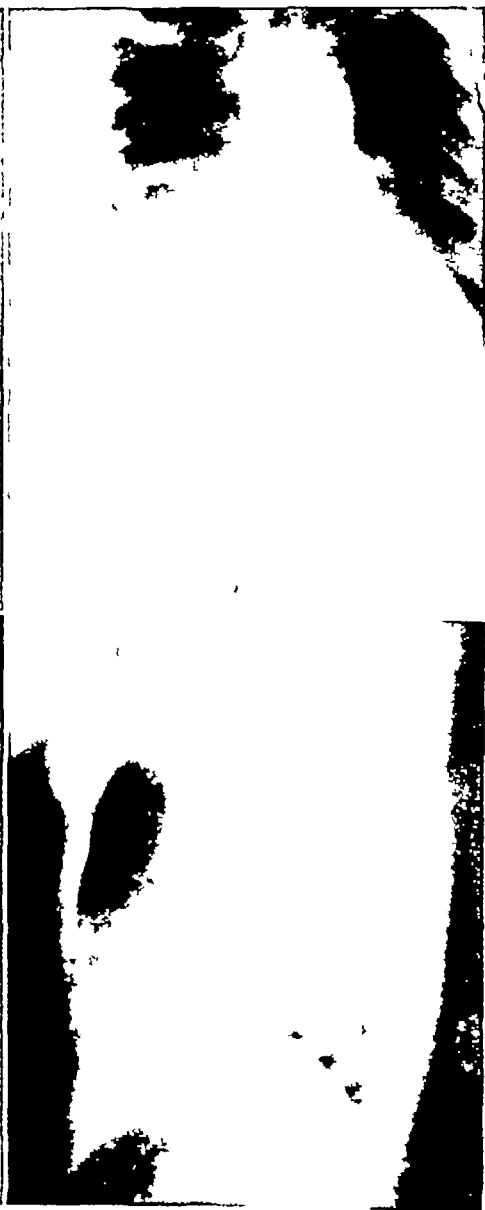


Fig 24

Fig 25

and torn soft tissues were excised, the torn kidney capsule was in view and the chest wall opening was large enough to insert the surgeon's hand and a metallic foreign body about 2 by $\frac{3}{4}$ by $\frac{3}{4}$ inches was removed from the right pleural space. The metallic foreign body was stuck on the outer surface of the right upper lobe. The pleural space was irrigated, two thoracotomy tubes were inserted in dependent positions, and the chest wall defect was closed by approximating the overlying soft tissues in layers. The patient made an uneventful recovery. Seven days later x ray pictures (Fig 25) showed complete lung reexpansion with only localized pleural thickening present.

This case falls into the group of modified thoracoabdominal explorations.

CASE 15—B L S, a 22 year old American corporal, was admitted twelve and one half hours after he was wounded. He sustained penetrating shell fragment wounds of the left forehead, left elbow, and upper abdomen. There was an entrance wound about 3 by 1 cm in diameter at about the seventh rib and midaxillary line on the left side. The abdomen was tender to palpation and rigid. Posteroanterior and lateral x ray pictures of the chest showed an irregular metallic foreign body near or at about the heart shadow (Figs 26 and 27). A laparotomy was performed through left paramedian incision. A tangential laceration about $1\frac{1}{2}$ inches long of the dome of the left diaphragm and a bleeding laceration about 2 inches long and 1 inch deep of the upper surface of the left lobe of the liver were found. The liver laceration was controlled by No. 1 chrome catgut mattress sutures, the hemoperitoneum was evacuated, and all the abdominal organs were inspected. Through the diaphragmatic tear it was not possible to palpate the metallic foreign body and since it was suspected to be near or at about the heart, the laparotomy incision was extended upward, the seventh, sixth, fifth, and fourth cartilages were divided, and the operative wound continued in the fourth intercostal space up to the midaxillary line. With the help of a self retaining retractor an excellent exposure of the heart was obtained. At about the apex a hematoma of the pericardium was found. The pericardium was opened and clotted blood was evacuated from the pericardial cavity. A perforation of the right ventricle about 2 cm long and $\frac{1}{2}$ cm wide was found. This wound was located near the septum in the neighborhood of the apex. While the patient received intravenous procaine and procaine was applied topically, through and through doubled 00000 braided silk sutures were placed into the heart muscle, traversing the laceration. The operator's left index finger was inserted into the right ventricle through the laceration, and the metallic foreign body was palpated and brought down to the area of the laceration. It could be removed only with an extension of the laceration about $\frac{1}{2}$ cm. After the metallic foreign body was extracted, the previously placed silk sutures were tied. During this procedure very little blood was lost. In about two or three minutes after the procedure was completed, the heart contractions slowed down and the ventricular contractions became weak and irregular. Cardiac massage was started immediately with resulting good heart action lasting only for about four to five contractions followed by a repetition of weakening of the ventricular contractions until almost ventricular standstill occurred. At this stage the cardiac massage was resumed with the same result. This vicious circle existed for two hours and forty five minutes, when cardiac massage resulted only in incoordinated muscle fiber contractions followed by complete cessation of the heart action.

This case represents one of the indications for abdominothoracic exploration.

DISCUSSION

In the management of these patients well tested conservative principles were applied. The value of repeated needle aspiration of hemothorax and hemopneumothorax is well established.²⁰ In addition, some changes in the indications for surgical interventions and some modifications of the methods of treatment of similar wounds during World War II were made.

The debridement and closure of sucking wounds of the chest and aspiration of the pleural space through the débrided wounds under general endotracheal anesthesia were found to be very effective. Only a few of the patients treated by this method needed subsequent needle aspirations. The value of this procedure was emphasized by Snyder² in 1945. The assurance of complete evacuation of the pleural space and full lung re-expansion at the time of the wound closure explains the good results.

The indications for open thoracotomy were few—seventeen in number, representing 12.9 per cent of all the chest injuries. In nine of them the chest was entered through an elective anterior incision (Diagram 1, *A, B, C, and D*). The idea of this technique was developed from an approach frequently used for elective lung resection.³ The magnitude of the thoracotomy was considerably lessened when the anterior approach was used. This proved to be the quickest method of entering the chest. The exposure was adequate and the wounds healed well. This was substantiated by follow-up reports.

Perforation of the diaphragm or the anatomic likelihood of such was not found to be an indication for formal thoracotomy. These patients were submitted to laparotomy and the diaphragmatic tear was closed from below as described previously. The likelihood of missing intra-abdominal injuries if the exploration is done through the chest is great. A thorough inspection of the intra-abdominal structures in such cases must be carried out. This cannot be accomplished as well through a thoracotomy wound as through a laparotomy incision, even if the thoracotomy incision is placed low and the diaphragm is divided.

The indications for exploring the chest as well as the abdomen in thoracoabdominal injuries are limited. Among the thirty-six thoracoabdominal injuries reported here, this was accomplished in thirteen or 36 per cent of the cases. In eight of these patients the anterior abdominothoracic approach previously described was used (Diagram 2, *A, B, C, and D*). This was a combination of a laparotomy with only the vertical part of the entire anterior thoracotomy incision which converted the thoracic and abdominal cavity into a single operative field and gave an excellent exposure. It provided easy access to parts of the abdominal cavity which could not be gained by separate approaches to the chest and abdomen. It proved to be the quickest way to expose the abdomen and the chest. The patient remained on his back throughout the procedure, which is the least disturbing position from the physiologic point of view. The wounds healed well and the costal arch became firm and bony. This was substantiated by follow-up reports.

In cases of acute emergencies such as war injuries the proposed anterior thoracoabdominal incisions were found to be most satisfactory in contrast to excellent thoracoabdominal approaches of Harper¹¹, Garlock¹², Carter¹³, Hargrove¹⁴, and others which were proposed primarily for elective condi-

SUMMARY

An analysis of 163 thoracic and thoracoabdominal injuries handled at a Mobile Army Surgical Hospital in Korea was presented. Twelve of these patients died, representing a mortality of 7.3 per cent.

Indications for surgical intervention were enumerated and the methods of treatment described.

A conservative attitude in the treatment of most of the chest injuries was emphasized.

The advantages of laparotomy in contrast to thoracotomy in the management of some of the thoracoabdominal injuries were stressed.

An anterior approach for thoracotomy and an approach for anterior abdominothoracic exploration were presented.

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MANAGEMENT OF WAR WOUNDS OF THE CHEST*

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ALTHOUGH the initial mortality of thoracic wounds sustained in combat is relatively high, the estimated delayed mortality experienced in the Korean conflict has been lower than that reported during the last two wars in which the United States has been involved. We have profited from the experiences of the physicians who served in the last war, and thus have a more complete understanding of chest physiology as related to derangements resulting from war wounds and greater knowledge of treatment of primary and secondary shock.^{1, 2, 6, 8, 9, 10, 11} We have new and more effective antibiotics, the Mobile Army Surgical Hospitals which give emergency treatment within a few miles of the front lines, and a more rapid system of medical evacuation, all which have done much toward decreasing the delayed deaths from chest wounds. Consequently, there has been greater opportunity for more definitive treatment of these patients with better final results.²¹

In this paper we make no attempt to give any over-all statistics concerning war wounds of the chest during this conflict, but only to analyze our experiences with the 1,535 chest casualties which were channeled through Tokyo Army Hospital between the beginning of the conflict and January, 1952. It is our understanding, however, that the majority of the chest casualties were sent to Tokyo Army Hospital.

The average chest casualty is seen first by the company aid man who applies an emergency dressing and is then evacuated by litter bearers, jeep ambulance, or helicopter to a battalion aid station where he may be given plasma or whole blood for replacement or to combat shock. Sedation is administered judiciously. The next step is movement to either a Mobile Army Surgical Hospital or an evacuation hospital by jeep, ambulance, or helicopter. Here emergency surgery, such as debridement and closure of the wound, is performed by surgical teams. Depending upon the patient's condition, he is moved on within forty-eight hours by ambulance, or by air to a field hospital or to a station hospital where he may remain for two to eight days. Following this he is transferred to a hospital ship or, more frequently, is flown to a general hospital in Japan. It should be noted that the system of evacuation is flexible enough to permit evacuation from the Mobile Army Surgical Hospital directly to general hospitals so that many patients arrive in Japan 48 hours after being wounded, a tribute to the system of air evacuation. However, most patients arrive at Tokyo Army Hospital within six days after being wounded. Some patients who were evacuated aboard hospital ships re-

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quired as long as thirty days en route. However, their treatment during this time paralleled treatment rendered at the general hospital. Definitive treatment is administered in the general hospital, and the patient is returned to duty or evacuated to the United States by air, depending upon his condition and the evacuation policy in force at the time. The patients from other United Nations countries are sent to their homelands.

In the early stages of the conflict, casualties could be hospitalized in Japan for only thirty days, thus making definitive treatment difficult to ascertain in many cases. However, as the bed capacity in the theater increased, the period of hospitalization was increased to 120 days, thus making definitive treatment of chest wounds practical.

It is worth mentioning that Tokyo Army Hospital is a United Nations installation staffed by United States military personnel. Patients were chiefly U S soldiers from all branches of service. We also cared for soldiers from all parts of the British Commonwealth, Turkey, Greece, France, Belgium, Thailand, Philippine Islands, Ethiopia, Colombia, and Puerto Rico. Most of the foregoing countries contributed nurses to the hospital staff, who also acted as interpreters. Despite their help, considerable language difficulties were encountered, thus complicating symptoms, diagnosis, and treatment.

The cases in this study do not include those consisting of superficial wounds of the chest wall, but deal primarily with wounds of the intrathoracic viscera solely, or in combination with wounds of the abdomen, nervous system, skeleton, etc.

War wounds, as a result of missiles propelled by explosives, may be classified as penetrating or perforating types. Penetrating wounds are those resulting from entrance of a missile with retention of that object as a foreign body regardless of the size of the wound of entrance. There is no wound of exit. Penetrating wounds are usually caused by mortar fire, artillery fire, hand grenades, land mines, small arms fire, in that order of frequency. Usually, the more severe wounds are of this type. Perforating wounds result from the through-and-through passage of a missile with a wound of entrance and a wound of exit. Approximately 90 per cent of the perforating wounds are caused by small arms fire, and the remaining 10 per cent by mortar and artillery fire, hand grenades, and land mines. In our series of cases, 1,007, or 66 per cent, of the wounds were of the penetrating type, 485, or 32 per cent, were of the perforating type, and 42, or 2 per cent, were results of crushing injuries.

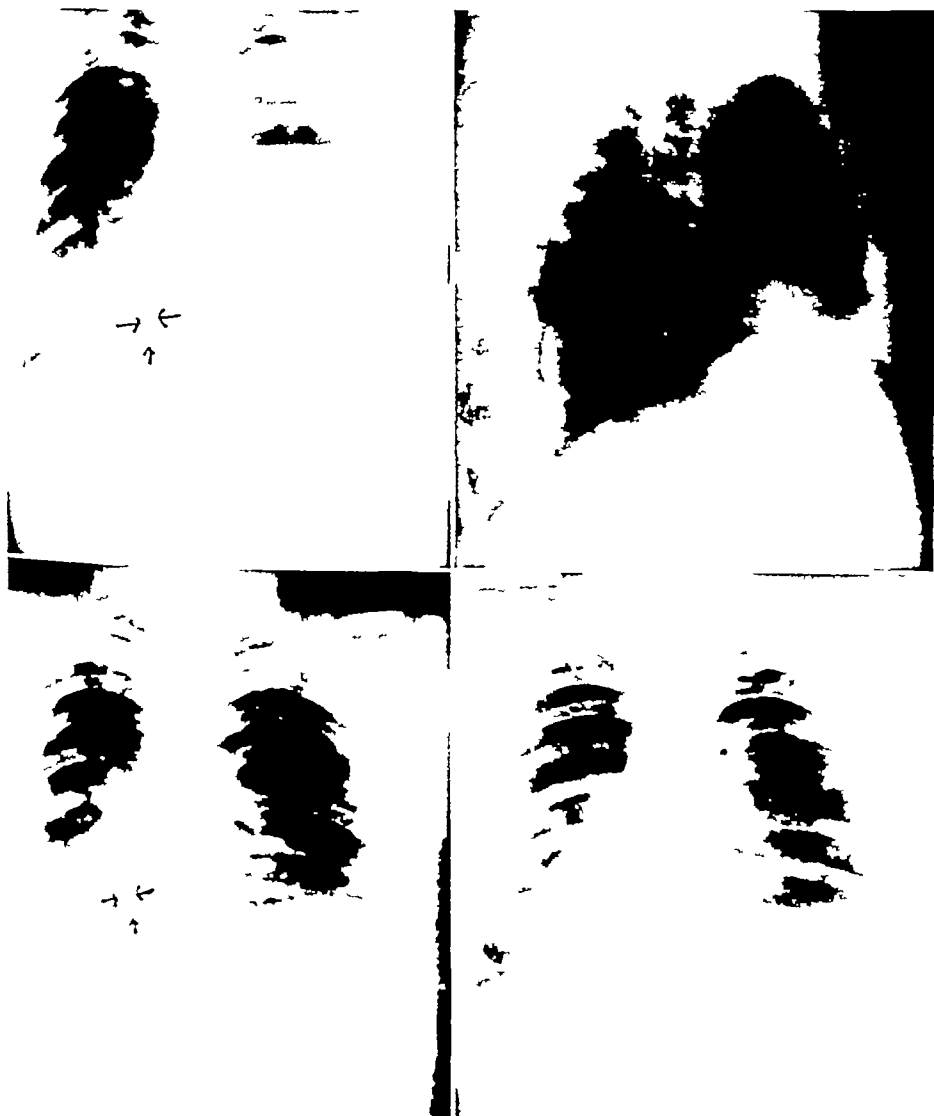
The restoration of normal respiratory and circulatory physiology is the prime factor in the management and treatment of thoracic wounds.

On admission to his hospital, each patient was screened by a medical officer, and all those requiring emergency treatment were seen immediately by the thoracic surgeon on duty who rendered the necessary treatment. Roentgenograms were taken immediately, when indicated. All patients had blood counts, urinalyses, and upright chest roentgenograms. They routinely received 300,000 units of penicillin twice daily, and, if indicated, $\frac{1}{2}$ Gm of streptomycin was administered with the same frequency. Patients were

placed in beds with the head elevated to a 45-degree angle and were encouraged to cough frequently. Sedation was kept at a minimum and, for severe pain, demerol was given in doses of 50 to 100 mg every four hours. In cases which had painful rib fractures intercostal nerve blocks with 2 per cent procaine hydrochloride anesthesia were done.

A

B



HEMOTHORAX

The most frequent complication of intrathoracic wounds is hemothorax with or without associated pneumothorax. This intrapleural blood may remain fluid or coagulate and begin to organize. It remains sterile, as a rule, but in some instances becomes infected. Of the total number of patients in our series, 952, or 62 per cent, had hemothoraces, either on admission or they developed them within the first two weeks after admission. Of this number, 704, or 74 per cent, remained sterile, and 248, or 26 per cent, became infected.

The policy here is to treat hemothorax by simple thoracentesis without an replacement. Localization of the fluid is important, since it may lie anteriorly, laterally, or posteriorly. However, it has been the experience at this hospital that it usually lies posteriorly. Aspiration is done with the patient in a sitting position, if his condition permits, under 2 per cent procaine hydrochloride local anesthesia. The skin is prepared with ether and tincture of merthiolate, and a skin wheal is raised over the most dependent portion of the fluid-filled space. The underlying muscles and pleura are infiltrated with procaine and a three-inch, 13- to 17-gauge needle with a short bevel is introduced. To this is attached a three-way stopcock and a 30 cc syringe. As much fluid as possible is removed, and the procedure is repeated every twenty-four hours until no fluid is obtained and the chest appears clear to physical and roentgenographic examination. Following removal of the fluid, 300,000 units of crystalline penicillin in 2 cc of saline is instilled. Specimens of the fluid removed are sent to the laboratory for culture and antibiotic sensitivity tests as indicated. The antibiotic of choice is administered systemically and intrapleurally, if infection is present, depending upon the manner in which the drug can be administered. Penicillin, streptomycin, terramycin, aureomycin, and Chloromycetin have been utilized.

Of the 952 patients with hemothoraces, 895, or 94 per cent, were treated with thoracenteses. The remaining 57 patients had minimal amounts of fluid which absorbed spontaneously. An average of 3.5 thoracenteses were performed on each patient, for a total of 3,082 thoracenteses. Several patients required only one aspiration, and one had twenty-five thoracenteses and ultimately required decortication.

Seven hundred fifty-five, or 79 per cent, of the patients with hemothorax completely recovered after being treated by thoracentesis and antibiotics alone. Six hundred fifty, or 68 per cent, were returned to duty. One hundred five patients were evacuated to the Zone of Interior because of other wounds, such as abdominal wounds, colostomies, central or peripheral nerve injuries, orthopedic injuries, etc., even though they were considered recovered as far as their chests were concerned. Forty-five patients had infected hemothoraces which were drained as a temporary procedure so the patients could be safely evacuated.

Many patients had clotted hemothoraces on admission to the hospital or within two days of being wounded. Apparently from a review of these

patients records, the hemothorax clotted within a few hours after wounding, because at no time was any fluid obtained by aspiration. In other patients, the hemothorax organized gradually, taking from three to four days to two weeks.

Decortication is the established method of treatment for these patients with clotted or organized hemothoraces.^{11 13 14 18} The first decortications were reported by Fowler in 1893¹¹ and Delorme in 1894.¹⁴ The procedure was revived during World War II, with good results by Samson and Britford and others. With the advent of penicillin even better results were obtained, and consequently many patients who would have been pulmonary cripples were restored to normal life.

In short, at this hospital the operation consists of a thoracotomy through a posterolateral incision under endotracheal gas oxygen-ether anesthesia following pentothal induction. A rib, usually the sixth or seventh, is resected, and a plane of cleavage is developed in the endothoracic fascia. As much thickened parietal pleura as possible is removed from the chest wall and diaphragm and all clot, debris and slough are evacuated from the thoracic cavity. The thickened cortex surrounding the collapsed lung is removed. The lung is re-expanded by positive pressure and any parenchymal tears are repaired. Foreign bodies, if present, may be removed during the operation. Two intercostal tubes are introduced, one at the level of the fourth interspace in the anterior axillary line, and the other in the eighth interspace in the midaxillary line. We use large stiff-walled right angle tubes which are attached to continuous low pressure suction. Roentgenograms are taken daily for the next four to five days. The tubes are removed within forty eight to seventy two hours.

Postoperatively, the patients are encouraged to cough frequently and are given deep breathing exercises. Approximately five days after surgery, the patients begin using a "blow bottle." We use the regulation waterseal drainage bottle filled three-fourths full with water. The patients inspire deeply and blow the expired air through the water in the bottle. About ten days postoperatively, the patients are gathered together for ten minutes every hour and are given deep breathing exercises. They are instructed how to breathe more deeply with the affected lung than with the unaffected one. To achieve this, the patients are shown how to place their hands over the anterior lower chest and breathe against their hands. In this way they themselves can feel that the normal side expands more than the injured one. Furthermore, they are given an exercise which causes one side of the chest to expand more than the other. The arm of the affected side is raised above the head and the whole body is bent toward the unaffected side during inspiration. During expiration, the arm is lowered and the body is returned to normal standing position.

We feel that the importance of these exercises cannot be overemphasized. They are important aids in the rehabilitation of decortication patients and,



Fig 2 —A Chest film showing organized left hemothorax with retained foreign body. B, Lateral film of same patient after decortication and removal of foreign body. C, Roentgenogram taken



Fig. 3. A, Chest film showing a large left diaphragmatic hernia accompanying perforation wound of the chest. B, Lateral film of same patient. C, Roentgenogram taken after repair of hernia.

for that matter, any patient who has had a thoracotomy. They help return the diaphragm and intercostal muscles to normal function and minimize the formation of adhesions in the costophrenic angle.

One hundred fifty-two decortications were performed, without a death, on patients with both infected and noninfected organized hemothoraces. Of these, 116, or 76 per cent, were infected and 36, or 24 per cent, were noninfected. Twenty patients in this group had thoracotomies of one kind or another performed in Korea and eight had decortications, which had been done prior to admission to our hospital. Of these, 134, or 88 per cent, were considered as having good results, and were returned to duty. Of the remaining 18 patients, 10, or 65 per cent, were considered to have fair results, and were returned to limited duty, and 8, or 55 per cent, were considered as having poor results.

After performing this operation at intervals of one week to eight weeks following injury, it was decided that the optimum time for decortication is within three to five weeks of injury. In those operations performed early, the bleeding was much more severe, there was more edema, and foreign bodies, if present, were more difficult to locate.

It is worth mentioning that 92 per cent of the patients who needed decortication had closed intercostal drainage tubes inserted in Korea. Their tubes had been kept in place for from two to eight days before the patient's admission to this hospital. Not infrequently the patients arrived at this hospital with the drainage bottles placed between their legs, or resting on their chests. Not always were they properly clamped off. Due to the negative pressure inside the chest, the fluid from the bottles was often sucked back into the chest and was another source of contamination.

It is our feeling that the best early treatment for hemothorax is thoracentesis. As has been stated, those patients who had intercostal tubes inserted in Korea made up 92 per cent of those who required decortication. In such cases, subsequent aspiration was more difficult and the incidence of infection was much greater.

We used streptokinase and streptodornase, as described by Tillett and Sherry,^{12 15 16} in 18 patients with hemothorax. One-half of these had infected hemothoraces, and the other half noninfected hemothoraces. The interval between the time of injury and the time the drugs were administered varied from four days to two weeks. Approximately 200,000 units of SK and 60,000 units of SD in 10 cc of distilled water were injected into the hemothorax. Eight to ten hours later the liquid portion of the hemothorax was aspirated and more streptokinase and streptodornase injected. This procedure was carried out for from four to ten days, depending upon the individual case. The drugs were injected at varied sites posteriorly, laterally, and anteriorly, and at different intercostal levels. All of our patients experienced toxic symptoms, such as high temperature, up to 105° F., general aches and pains, and occasional vomiting. Daily chest roentgenograms generally failed to show any marked change except in one case. In this patient,



FIG. 8. A, Chest film showing left organized hemothorax with retained large metallic foreign body. B, Natural film of same patient. C, Chest film taken following decortication and removal of foreign body.

while the drugs dissolved some of the adhesions and the lung partially collapsed, they failed to dissolve the thickened pleura. In view of the results obtained, the use of these drugs was discontinued.

One death occurred in this group of patients with hemothorax. A patient with an empyema died of pulmonary embolism.

FOREIGN BODIES

As is well known, during war chest surgery, numerous foreign bodies are encountered in the chest wall, the pleural space, in the lung parenchyma, or mediastinum. Most of these are a result of penetrating wounds. In this hospital only those foreign bodies which exceeded 15 centimeters are removed, unless, of course, they are in such a location as to be regarded as dangerous to the patient or give rise to some pathologic change within the chest. Smaller foreign bodies in the region of the heart and great vessels and in the pulmonary parenchyma are observed carefully by repeated roentgenographic and electrocardiographic examinations for at least one month. If there is no evidence of reaction about the object, and the patient's condition is satisfactory, these foreign bodies are not disturbed. Persistence or development of reaction about the missile indicates the need for exploration and removal.

Of our patients, 104 had retained foreign bodies. Thirty-eight were in the chest wall accompanied by intrathoracic damage and rib fractures. Thirty-five were in the lung parenchyma, and thirty-one were in the pleural space and were removed at decortication. Eighty-five per cent of the metallic foreign bodies were shell fragments which varied in size from 1 cm. to 8 cm. in diameter. Most of these were irregular in shape with ragged edges. The remaining 15 per cent were bullets of different caliber. The incidence of infection when the foreign body was a shell fragment was quite high, being from 60 to 70 per cent. When the foreign body was a bullet, the incidence of infection was about 10 per cent.

The thirty-eight foreign bodies located in the chest wall, which are mentioned here, were those cases in which the foreign body was so located as to require thoracotomy for removal. We removed numerous metallic foreign bodies more superficially situated in the chest wall by simple procedures, and do not have figures concerning these. All of the patients in this group returned to duty.

The removal of intrathoracic foreign bodies is accomplished by open thoracotomy through a posterolateral incision with resection of a rib. An incision is made in the lung surface nearest to the object and the missile is removed. Infected lung tissue is removed by wedge or segmental resection. Twenty of these patients had partial lobectomies. All 35 of them returned to duty.

Thirty-one foreign bodies were removed at the time of decortication. Twelve of these also suffered some damage to the lung parenchyma and needed

partial resections at the same time. All of these patients returned to full duty. The foreign bodies removed from the pleural cavity consisted not only of shell fragments and bullets, but pieces of clothing, fountain pens, and in one case an olive.

Of the 37 patients admitted to this hospital who had thoracotomies performed in forward areas, 23 were thoracotomized apparently solely for the removal of a foreign body. Although the patients ultimately recovered, we considered 35 per cent of them to have a poor result because recovery was slower and the amount of fibrosis present was greater, thus diminishing pulmonary function. Eight of these patients developed empyema.

In our series, the 73 patients who were thoracotomized solely for the removal of a foreign body all recovered uneventfully and returned to duty.

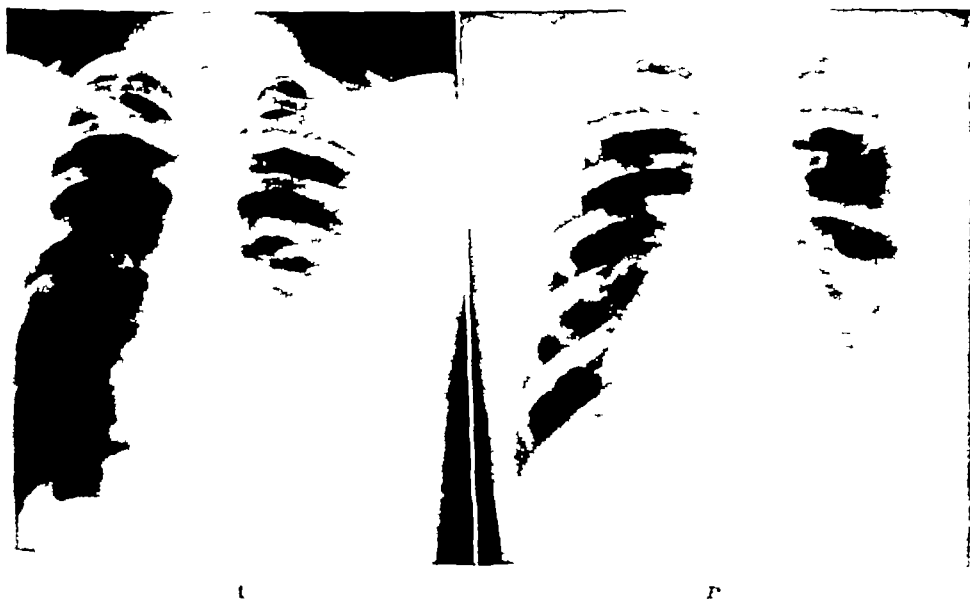


FIGURE 1. *A* Chest film showing organizing right hemothorax. *B* Roentgenogram taken following decortication.

In view of the good results obtained by leaving the foreign body in at all possible undisturbed for two to three weeks we feel that nothing is gained by early thoracotomy for removal of foreign bodies. Delaying operation until the inflammatory reaction has subsided obviates considerable unnecessary bleeding at operation and makes location of the foreign body easier. Also the patient is usually in much better condition to stand the operation.

MEDIASTINAL WOUNDS

Forty-two, or 2 per cent, of our patients suffered mediastinal wounds with retention of foreign bodies. The small number of these cases admitted to this hospital is probably accounted for by the high initial mortality of this type of injury. Twenty-two of these were in the pericardium with effusion and four were in the mediastinum and four were in the heart muscle.

The cases with pericardial effusion had very small metallic foreign bodies in or about the pericardium. The effusion usually developed suddenly, sometimes one to six weeks after injury, at which time the patient experienced severe precordial pain, rise in temperature, tachycardia and shortness of breath. The heart shadow was enlarged on roentgenogram and the electrocardiogram showed pericardial changes. These patients were observed very carefully with frequent roentgenograms, electrocardiographs, and physical examinations. Most of these patients had pericardiocenteses. Some required only one and some as many as ten. In most of these cases pyogenic organisms were obtained from culture of the fluid removed at the first or second aspiration. Sensitivity tests were done routinely and the chemotherapy of choice was administered both systemically and locally. Treatment was continued until the roentgenographic, physical, and electrocardiographic examinations were normal. All of these patients recovered and were evacuated to the Zone of Interior.

Sixteen patients had foreign bodies in the mediastinum. All of these were removed through a thoracotomy. Four patients had mediastinal abscesses about the foreign bodies. These were débrided and the abscess cavity left open into the pleural space which was drained in the routine manner. Ten of these patients returned to duty, and the remaining six were evacuated to the Zone of Interior.

Four of these patients had bullets of various caliber in the myocardium. In none of these cases did the bullet completely perforate the heart wall. The bullets were removed through a left thoracotomy. Prior to removal, several U-shaped sutures of heavy silk were placed around the missile to control any bleeding which might occur and to reinforce the damaged heart wall. These were tightened and tied when the missile was removed. All of these patients recovered and were sent to the Zone of Interior.

Except for the ten patients who went to duty in this command, all patients with heart, mediastinal, and pericardial injuries were sent to the Zone of Interior for duty because it was not felt advisable to subject them to the rigors of combat duty.

One patient, who had a small metallic foreign body in the myocardium, succumbed to cardiac tamponade twelve hours after admission before operation could be performed.

One thoracotomy was performed to remove a partial denture which was lodged in the lower third of the esophagus. The patient had swallowed it when he was blown from his foxhole by artillery fire. He made an uneventful recovery and returned to duty.

Another case of interest is one in which a 22 caliber bullet perforated the left upper chest and superior mediastinum and entered the aorta. The right leg became gangrenous twelve days after injury and was amputated upon admission to this hospital. The bullet was found in the lower third of the femoral artery.

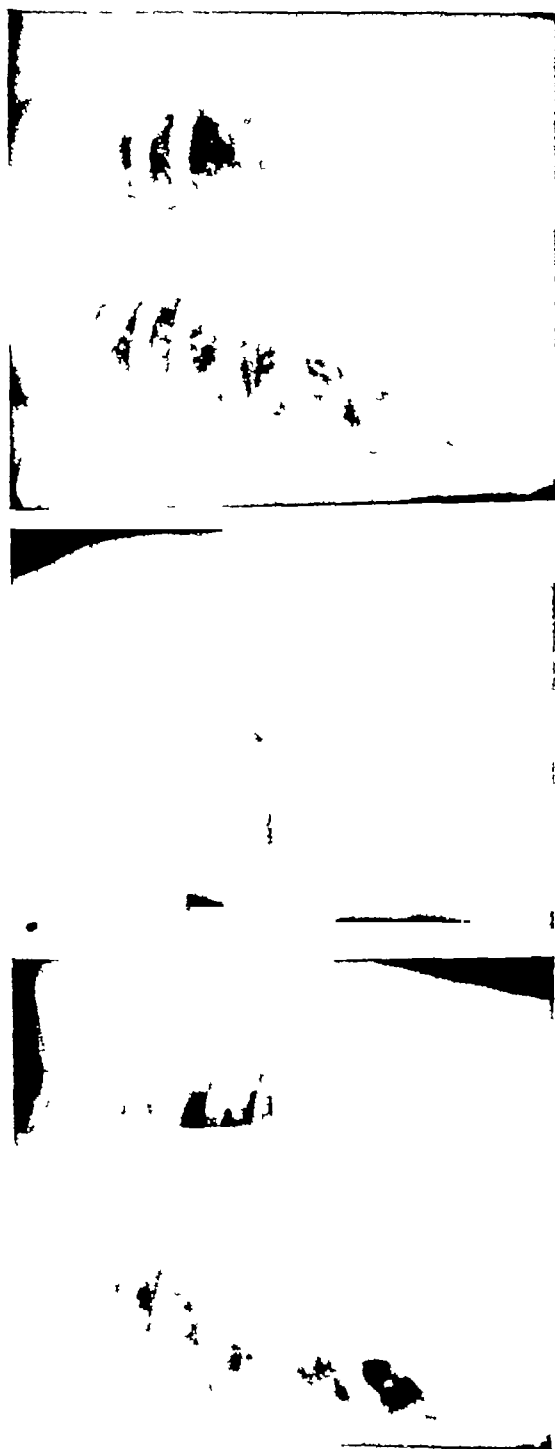


FIG. 1. The three films showing, partially collapsed right lung with hydropneumothorax which subsequently became infected B, Internal film of same patient C Roentgenogram taken following decortication

PNEUMONITIS AND ATELECTASIS

Two hundred sixty-one, or 17 per cent, of our patients had pneumonitis and/or atelectasis. This is primarily a diagnosis resulting from clinical and roentgenographic examination. There is a decrease in breath sounds over the area of the lung involved with slight dullness to percussion. Occasionally moist râles will be present. Roentgenogram reveals an area of increased density, not conforming to any segmental pattern and indistinguishable from a patch of consolidation. There was evidence of intraparenchymal hemorrhage, and invariably the center of density represented the passage of a perforating missile, a retained foreign body, or lung underlying fractured ribs. Treatment of this condition consisted of parenteral administration of antibiotics, frequent coughing, and ambulation, if the patient's general condition permitted. Retained foreign bodies were treated as has already been described.

In atelectasis, bronchoscopy has been the treatment of choice. The pneumonitis generally has cleared under the above treatment in within about three weeks.

PNEUMOTHORAX

Twenty-three patients in this series were admitted with pneumothorax alone. Treatment consisted of removal of air by needle aspiration and checking intrapleural pressures initially and before withdrawing the needle. Following each thoracentesis, 300,000 units of penicillin were instilled. In six instances, the lung failed to expand on this treatment and it was necessary to perform a closed thoracotomy drainage with an intercostal catheter with suction in an attempt to accomplish expansion.

No cases of tension pneumothorax were seen.

LUNG ABSCESS

Nine patients were admitted with lung abscesses. Three followed crushing injuries to the chest which were sustained in jeep and truck accidents. Six followed penetrating or perforating wounds of the chest. These were treated by pulmonary resection, six by partial lobectomy and three by lobectomy. Partial thoracoplasties were performed on two patients to obliterate the space left by upper lobectomies. All patients recovered uneventfully, five returned to duty, and four were sent to the Zone of Interior because of other injuries.

RIB FRACTURES

Three hundred fifty-four patients had fractured ribs. Most of these accompanied perforating or penetrating wounds, but 42 were the results of crushing injuries to the chest. The majority were comminuted fractures with bone fragments often driven into the pleural space and lung parenchyma. Many of these were removed at thoracotomy.

One of the most frequent complications was hydropneumothorax. In several instances, the lung was successfully re-expanded by thoracentesis only to collapse again. On closer roentgenographic examination, a bone fragment

or fragments were seen to project into the chest and would puncture the lung each time it re expanded. In these cases, segments of the fractured ribs were removed and the edges trimmed. The lung then re expanded and remained so.

When more than two ribs in one hemithorax were fractured, they were immobilized in the usual manner with adhesive tape.

THORACO ABDOMINAL WOUNDS

One hundred eight patients suffered wounds to the diaphragm. These injuries were repaired at the time of thoracotomy. The wound was débrided, the edges of the diaphragm trimmed and approximated with interrupted sutures of 00 silk. Those patients who suffered damage to the diaphragm only in conjunction with the chest wound were usually returned to duty.



A — Chest film showing original left hemithorax. B — Postoperative film taken after debridement.

In the event the wound was on the right side and there was also liver injury, the liver wound was débrided and cleaned at the same time. If there was infection present, the liver was drained with a Penrose drain which was positioned laterally below the diaphragm. Also foreign bodies, if present, were removed.

In two cases with perforating wounds of the left lower chest and upper abdomen the patients were admitted with the stomach, spleen, and colon in the chest. The lung was completely collapsed and the mediastinum was shifted to the right. In both cases the stomach was perforated several times. At operation the stomach was repaired, the abdominal contents were returned to their normal positions, and the diaphragm was sutured. All patients made uneventful recoveries and returned to duty.

The following chart shows the extent of abdominal wounding in combination with thoracic wounds encountered in this hospital

Liver	116
Spleen	60
Large intestine with colostomy	44
Small intestine	10
Stomach	32
Kidney	20

The majority of the patients with spleen injuries underwent splenectomies in forward areas and were sent here for repair of their chest wounds. Generally, in those cases in which the patients suffered only spleen injuries, we carried out definitive chest surgery and they were returned to duty.

Patients with serious liver infections, colostomies, nephrectomies, multiple intestinal perforations, with or without resection, etc., usually were evacuated to the Zone of Interior. Most of these patients were in poor general condition, and only the temporizing procedure of open drainage with rib resection was carried out so that they could be safely evacuated. There seemed to be no point in subjecting these patients to definitive chest surgery in the theater, since few of them would be able to return to duty because of their abdominal wounds.

In our experience, many cases with thoraco abdominal wounds involving the upper abdomen could be handled adequately through the thoracotomy. The exposure is good, often better than with the abdominal approach, and it obviates the necessity of an abdominal or thoracoabdominal incision. It is our feeling that those patients who need more extensive abdominal surgery should have separate thoracotomies and laparotomies rather than the thoraco abdominal incision. Infection, if it develops, is more localized, and the patients seem to make better progress.

Three deaths occurred in this group. One patient died seventy-two hours after closure of his disrupted abdominal incision. Two deaths were due to generalized peritonitis in patients with numerous intestinal perforations and extensive liver damage.

NERVE INJURIES

One hundred ninety-seven of our patients also had nerve injuries. Of these, 97 were peripheral nerve injuries, 86 were paraplegics, and 14 were intracranial.

The general principles of treatment were the same for handling all these types of injuries. However, there was considerable difficulty in adequately aspirating or draining the pleural fluid in the treatment of paraplegics because of the horizontal position required on the Stryker frame. In addition, the ability to cough was usually lessened because of the paralysis of the abdominal musculature and the position of the patient.

All of these patients received only palliative treatment: thoracentesis, or thoracotomy drainage with rib resection and were evacuated to the Zone of Interior.

One death occurred in this group. A paraplegic with a chest wound showed signs of increasing intracranial pressure and died following decompression by burr holes.

ORTHOPEDIC INJURIES

Four hundred eight patients had serious orthopedic problems. Most of them had fractures of the extremities. The majority of these patients received only temporary treatment and were evacuated to the Zone of Interior. However, some recovered from both their chest and orthopedic injuries and returned to limited duty in the theater.

SUMMARY

During the Korean conflict, between July, 1950, and January, 1952, 1,535 chest casualties were admitted to Tokyo Army Hospital. Of these, 66 per cent had penetrating wounds, 32 per cent had perforating wounds, and 2 per cent were crushing injuries of the chest.

Nine hundred fifty-two patients developed hemothorax, 74 per cent of these remained sterile, and 26 per cent became infected. Sixty-eight per cent of these patients were treated by thoracentesis and antibiotics alone and recovered and returned to duty. One hundred fifty-two decortications were performed without a death. Eighty-eight per cent of these patients were considered as having good results and returned to duty. 65 per cent had fair results and returned to limited duty, and 55 per cent were considered to have poor results and were sent to the Zone of Interior for further treatment. Ninety-two per cent of the patients who required decortication had closed intercostal tubes inserted in Korea. In view of the fact that 79 per cent of the patients with hemothorax recovered when treated by thoracentesis and antibiotics we feel that thoracentesis is the best early treatment for hemothorax and that if this more conservative treatment had been carried out more frequently instead of closed thoracotomy drainage perhaps decortication could have been avoided in some cases.

One hundred four patients had foreign bodies removed from their chests. Seventy-three thoracotomies for removal of foreign bodies were performed. Twenty of these patients also had partial lobectomies. All recovered uneventfully and returned to duty. Thirty-one patients had foreign bodies removed at decortication. All 104 patients returned to duty. Twenty-six patients were thoracotomized in forward areas for removal of foreign bodies. These had either fair or poor results because of increased morbidity and delayed pulmonary function. When compared with the results obtained at delayed removal it would seem that it is best to leave the foreign body undisturbed if possible for later removal.

Nine patients who had lung abscesses were treated by lobectomy or partial thoracotomy and recovered and returned to duty.

One hundred eight patients suffered injuries to the diaphragm. These were treated at thoracotomy. One hundred seventy-four patients had thoracic wounds. In general these patients with serious liver infections

colostomies, multiple intestinal perforations, etc., were treated conservatively by open thoracotomy drainage with rib resection so that they might be safely evacuated to the Zone of Interior

The 197 patients with serious nerve injuries were treated with thoracotomy or rib resection and thoracotomy drainage and then evacuated to the Zone of Interior. The 408 orthopedic injuries were treated much in the same manner.

Our operative mortality at this hospital was 0 per cent. The overall mortality was 0.4 per cent.

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DISCUSSION ON "THE CARE OF THORACIC AND THORACOABDOMINAL WOUNDS IN THE COMBAT ZONE IN KOREA," BY MAJOR EDWARD ERNEST ROCKEY, AND "WAR WOUNDS OF THE CHEST: EXPERIENCES WITH 1535 KOREAN CASUALTIES IN AN ARMY HOSPITAL OVERSEAS," BY MAJOR A. R. VALLE

DR. HOWARD E. SNYDER, Winfield, Kan.—I think Dr. Berry's fine address clarified some of the statistics you have just heard, but it might be well to call attention to

Our deficiencies in Korea. You will note that in this series of chest cases the error of emphysema has again risen to 25 or 26 per cent. That is probably just a poor fault as an conscious citizens as the fault of any individual surgeon. Dr. Linn has pointed out that the work has done with these mesh units, one per division. They did not only the job they were supposed to do, the care of first priority casualties, but earned for all the rest of the casualties as well. They had little time for the so-called minor wounds. The terrific casualty loads in September and December of the first year of the war were not expected. There were just too many to be properly taken care of by the hospitals available. That is one reason for the rise in the emphysema rate. There are other reasons and, again, we ourselves may be deficient. The Army did not have a great number of specialists, fully trained men. The men who were there did a good job and they could with their qualifications and with the time they had available. There were very few men with Board qualifications in general surgery and, probably with qualifications in chest surgery in Korea in the early months of the war. We certainly are in part responsible for that because the Army is limited now in the men they can call. Class I and Class II in the doctor draft classification do not provide many specialists.

I was rather surprised at Major Hockey's conclusion that an abdominal incision was the best in handling a wound of the diaphragm with certain exceptions. I think in World War II we formed the other conclusion, most of us who liked to do chest surgery almost never surgery did before the war was over; would much rather approach a diaphragmatic wound from the chest and we preferred the conventional posterolateral incision in most cases unless the wound was such as to indicate an interior incision. We liked to do the vertical incision through the cartilages except in certain specific cases. There were those of us who did like the combined thoracodorsal incision, in which a small extension of the thoracotomy wound into the anterior abdominal wall. But I cannot all decided exploration in the abdomen. There were others who, as Major Valle expressed himself in the second paper preferred separate incisions. I think it is preferable to use the thoracotomy incision and close the diaphragm before the abdomen is opened. I believe a diaphragmatic wound unsutured may become a sucking wound as well as the abdominal incision is opened with disastrous results. If the diaphragm is well as the wall is repaired before the abdomen is opened that sort of catastrophe can be

But I would like to say in Antonio—I should like to congratulate the author on the completion of their work. I as well as many others here, know full well the difficulties of trying to end a report out of a combat or a particularly in the forward area. It is certainly to be commended for their evaluation of results for a hernia that now where from 25 to 30 per cent of this type of wound may have major results. I would like the evaluation of results of this type of wound particularly difficult. I still like to make it very clear in the informal remarks that I sound at all and in the Surgeon General should be identical read this discussion. I would like to know that I could not do any better. I have no doubt what I cover to end up

be qualified, such a statement quoted out of text would in my experience be quite in error. Someone will assuredly quote it out of text without the explanatory qualifications, and trouble would undoubtedly result. I feel there is no argument if someone wants to operate through the abdomen and handle the involved diaphragm or through the chest or through a combined approach. That is a matter of personal experience, but I think it is dangerous to have this get into print without a careful explanation, because involvement of or suspicion of involvement of the left diaphragm is an absolute indication for exploration, in my experience.

On my return from the Mediterranean Theater in World War II, I became interested in the mortality and morbidity of thoracoabdominal wounds in civilian hospitals, and I became aware of the fact that the mortality was twice what we had in field hospitals. The majority of factors involved are on the side of a lower civilian mortality. The time interval between wounding and definitive surgery may average as high as eleven or twelve hours in war wounds, whereas it is a matter of minutes or a few hours in civilian wounds. There is not the gross contamination in civilian wounds that is constantly experienced in war wounds. Furthermore, it is relatively rare to have as high a percentage of major associated wounds in civilian practice as one has in war wounds.

On the other hand, in war time one is operating on young, previously healthy individuals, and it is also true that because of the promptness with which civilian wounds are carried to the hospitals, more mortally wounded might therefore be seen than would arrive at field hospitals. However, these last two factors do not negate the previous ones, and when one considers that the surgery of trauma is simplicity itself—one only stops a hemorrhage, cuts something out, exteriorizes it, or sews it up—the cause of the difference in mortality must therefore be searched for elsewhere.

I think that the big difference lies in one particular factor, that is, preoperative management or, what should be better termed, the resuscitative phase. I do not believe that the information we have obtained concerning the importance of this phase has been carried over sufficiently into civilian life. The idea of spending two or three hours resuscitating a patient before operating, and not taking him directly to the operating room simply because he has a sucking wound, is not well enough known. Furthermore, many cases, if properly resuscitated, will not require surgery, for one can evaluate only the necessity for surgery after one notes how a patient reacts to adequate resuscitative measures, and I think one need only mention that a patient properly resuscitated will stand surgical interference far better, even if in some cases the surgical interference is not warranted. I must admit honestly and many of you men who did similar work in the Second World War, if you are honest with yourselves, will admit to plenty of cases that you opened, and after you got in you wondered what you were doing in there. Personally, that experience decreased from Sicily to Southern Italy to Northern Italy, etc., for me. As I saw more and more cases, I was actually operating fewer of them but spending more and more time in the shock tent resuscitating the patients. After they have been resuscitated properly, then and only then can you properly evaluate them. Surgical insults that occur otherwise are inexcusable, and certainly they are inexcusable in civilian life where one does not have the pressure of 20 more cases waiting in the shock tent.

DR. FREDERICK R. HARPER, Denver—I wanted to discuss these papers only to agree with the authors. During the Second World War, I was in the South Pacific in a general hospital. I thought we had a rather unique experience in that we received two groups of patients. One group came from Saipan and Peleliu. Those were campaigns where the medical corps was hard pressed and many of the wounded were taken directly to the beaches and put on hospital ships and sent down to us. The Navy surgeons in those hospital ships were very hard pressed so they did very little for the ordinary thoracic wound. The other group of patients came down from Leyte, through field hospitals, evacuation hospitals, and so on. These patients had been treated before they got to us. Tubes had been used in most cases. There was a very marked difference between the two groups, the group that came down in Navy ships, who had had practically no treat-

best, did much better than the group that came down through the hospitals and had tubes and drainage. There were fewer decortications necessary and fewer complications in the untreated group. We could not help noting that patients who had needle aspiration only did much better than those who had tube drainage. I see no indication for tube drainage unless you are dealing with a positive pressure pneumothorax, otherwise needle aspiration is adequate.

The indications for immediate open thoracotomy are few: (1) large open sucking wound; (2) bleeding which cannot be controlled because it is coming from a major vessel; (3) pneumothorax which cannot be controlled because it is due to an open bronchus; (4) hemothorax tamponade; (5) some patients with flail chest, I think, can be handled better by open thoracotomy; and (6) the thoracoabdominal wounds. My only point of disagreement with the authors with regard to thoracoabdominal wounds is if I understood correctly, is that I believe many patients who have thoracoabdominal wounds should have the thoracotomy done first or at the same time, even though a thoracotomy would otherwise not be necessary. If you do not stabilize the cardiorespiratory system before you open the abdomen, I think you are inviting difficulties.

DR LAURENCE MISCALL, New York—The experience from which I speak is that seen in a large thoracic center in the rear area in England. Here Dr Harrison and I had an excellent opportunity to see the results of treatment that was carried out in all the prior stations, including station and general hospitals in England. I agree certainly with the statement that most hemothoraces can be treated effectively by aspiration and thoracocentesis measures. In a survey of over 200 patients, we found that at least 90 per cent responded favorably to these methods. However, in the hemothorax or hemo-pneumothorax which could not be controlled readily by repeated aspirations, good results followed closed drainage which was maintained long enough to permit expansion of the lung. We found poor results when the drainage was ineffective or used for too short a time. These individuals had considerable trouble with clotting and infection before or after evacuation to the rear. This is a sound basis for questioning the advisability of the early removal of the tubes after thoracic exploration in the forward area.

In thoracoabdominal wounds we found that the patients who had difficulty had either one or these therapeutic deficiencies, the abdomen and chest had not been separately repaired by complete repair of the diaphragmatic defect, the chest had not been adequately drained, exploration had been inadequate, defects below the diaphragm had been neglected frequently. This was particularly true on the right side where unsuspected lesions of the liver had caused many patients to return to the hospital with pleural and diaphragmatic infection. These facts prompted us to explore adequately both above and below the diaphragm as a routine.

The other point that might be suggested to the individuals in Korea concerns the hemothorax with unavoidable delay in treatment. The use of streptokinase and heparin immediately after operative decortication has been quite satisfactory in results.

DR GEORGE N. J. SOMMER, Jr., Trenton, N. J.—Chiefly, I wish to agree with all working in the quiet atmosphere of a thoracic surgery center in England. I shall never through examination of the patients and their records to decide whether aspiration or tube drainage was the best method of preventing clotted hemothorax. I had no heart-cut opinion. Furthermore, I have seen patients in civilian hospitals who had extensive clotted hemothoraces within twenty-four hours after being stabilized by closed drainage by intercostal tubes would have prevented clotting in these hemothoraces if aspiration and the clotted hemothorax had been corrected immediately. I am sure that the judgment of those who are treating patients in the forward area is correct. Certainly the hemothorax in the forward area should be corrected by aspiration and the clotted hemothorax after a rest rather than repeated

aspiration simply because he may not have time for the latter. For myself, I wish to state that I feel intercostal drainage is properly used following operations for injuries to the chest and lungs.

Experience in the thoracic center did show, however, that the best results in the treatment of thoracic injuries were obtained by well trained surgeons. The caliber of treatment of patients admitted to the center depended upon the skill and experience of the men giving the primary treatment near the front lines.

The suggestion that an upper abdominal incision be carried up over the anterior thorax with transverse division of ribs or cartilages to afford exposure of the intrathoracic contents is a poor one. The division of multiple ribs in such a manner will provide an immediately unstable thoracic wall with the increased hazard of atelectasis and paradoxical movement. To wire such ribs to avoid the unstable thoracic wall will undesirably prolong the operation time. Furthermore, late thoracic deformity will occur unless very meticulous care is used to prevent it. Even the best of late care will not always prevent deformity with multiple rib fractures or division. If thoracic exploration is required after abdominal operation, the incision could be prolonged upward in the standard method through an interspace or another incision could be made.

DR WARRINER WOODRUFF, Saranac Lake, N. Y.—As I listened to these two papers, it seemed to me that they present experiences of two distinct and separate types. One is that of a surgeon in the forward area who is extremely hard pressed with many casualties and insufficient time to give to them, and the second paper reflects the problem as seen in a rear area hospital in Japan. I think the figures show the difference. My experience was limited to two campaigns: Iwo Jima and Okinawa. I was on Guam during both campaigns. From Iwo Jima many of our patients were flown in 650 miles, and we had one boy admitted ten hours after he had been hit. In general, casualties from Iwo Jima were seen earlier than those from Okinawa, because policy of evacuation was changed and it became practice not to transport chest cases by air, so we received almost no casualties from Okinawa for primary treatment. The reason I mention that is this: I know that our incidence of empyemas increased, the longer the interval between wounding and the time we saw them. I don't know what the experience of others has been, but it seems to me that in the long run it may be better to lose an occasional chest case by air transport than to take the hazards of the necessary delay of water transport. If I have understood these figures correctly, the average time before admission to the hospital in Japan was six days, but on some patients there was a delay of up to thirty days. I wonder if complications were not higher in this latter group. Certainly we found it so.

There is just one thing I should like to say, if we are helpful to Dr. Rokey. There is one patient whom he cited who had a crushing injury of the chest with bilateral pulmonary involvement, who died very soon postoperatively, coughing and raising blood. As he stated, they were unable to keep the airways free despite all attempts, including bronchoscopy. All right—in a forward area such as that, you just do not have time to do the amount of tracheal aspiration that should be done, and I wonder if it would not be worth while to call Dr. Rokey's attention to the possibility of tracheotomy on some of these severe cases, so that the nurses may keep the secretions aspirated regardless of the patient's condition.

DR MARK H. WILLIAMS, Binghamton, N. Y.—I want to say one word about the management of hemothorax. It is well established that needle aspiration is usually an effectual measure, but if aspiration is incompletely done, pleural complications necessitating drainage or decortication may occur. A simple and effectual way of performing thorough aspiration has been helpful to me, and I think it should be applicable to military practice. This is actually a minor operating room procedure which requires a strong source of suction. One positions the patient in the supine position on the operating table, elevating the head and shoulders to about 45 degrees and then introduces a trocar through the first or second interspace. The Corvillo's thoroscope trocar, which permits the introduction of the 16 gauge urethral catheter, has been used. The catheter will pass back

Also, it often gives a false sense of security in that drainage is instituted and then for gotten about, because of the feeling that the problem has been solved. However, it is later that the consequences must be dealt with.

Regarding Dr Woodruff's comment about the average time elapsed between injury and date of arrival at the general hospital of six to thirty days, respectively, in our latter group the treatment received on the hospital ship paralleled that received in the general hospital.

MAJOR ROCKEY—I would like to express my thanks to Dr Snyder, Dr Shefts, Dr Harper, Dr Miscall, Dr Sommer, Dr Woodruff, Dr Williams, and Dr Sarot for their very valuable remarks which were of great help in re-emphasizing many of the important points of this presentation.

As Dr Woodruff very well stated, the experiences in the forward area are distinctly different from the ones in the rear. The more thorough and understanding care is given at a Mobile Army Surgical Hospital level to these patients, the less complications requiring elective surgical interventions are faced in Japan. As Dr Snyder indicated, this can be accomplished only if more trained surgeons are available in the combat zone, or perhaps the men in such units could have intensive on the spot training in the early and intelligent management of thoracic and thoracoabdominal injuries.

The reason for the difference in the statistics between the forward area and the cases handled in Japan is obvious. When a Mobile Army Surgical Hospital is quite near to the front line with the helicopters, patients reach us who would otherwise be dead on arrival. As a matter of fact, a number of them were dead by the time we put them on the operating table for resuscitation purposes, and we included them among our operated cases. Most of the other cases were of great risk, and this is the reason they were operated on in a forward unit.

It is a great regret to me that I was not able to be present at the meeting of this Association, particularly since, on one of the main issues of my presentation, the management of diaphragmatic perforations, I could have elaborated in greater detail. In response to the remarks of Drs Snyder, Shefts, and Harper, I wish to say that it is not a matter of personal preference that I suggest laparotomy for the management of thoracoabdominal wounds, but it is the question of doing the least number of and the least extensive procedures which will provide an opportunity to re-establish as normal cardiorespiratory physiology as possible as well as correction of the intraabdominal damage. This choice is not a question of taking the attitude of the general surgeon who is, perhaps, not too well versed in the various thoracic approaches, since I am primarily a thoracic surgeon. In view of the fact that the diaphragmatic tear is taken care of as a first step in the operation after the abdomen is opened, its sucking effect is eliminated, the pleural space is evacuated, and the lung is fully expanded. This can be accomplished with ease and without danger since the patient is handled under endotracheal anesthesia. Under these conditions, no ill effects of this procedure were observed. The majority of the thoracoabdominal injuries do not require exploration of the chest, just as the majority of the chest injuries can be taken care of by aspiration only. In other words, we are saving a thoracotomy or the thoracotomy part of the abdominothoracic exploration without sacrificing complete evacuation of the pleural space and full lung re-expansion prior to the diaphragmatic closure. One should not hesitate to extend the laparotomy incision into an abdominothoracic one if difficulties are encountered either in the management of the intrathoracic phase of the injury or in the closure of the diaphragmatic perforation. In my experience, in the majority of the cases, such extension was not necessary. Had I handled these patients by thoracotomy first, many of the patients who were submitted only to laparotomy would have been submitted to thoracotomy as well or to combined thoracoabdominal exploration without accomplishing more than was accomplished by the method I propose. Among the casualties, there were several cases of perforation of both leaves of the diaphragm, which were successfully handled by laparotomy, evacuation of the pleural spaces through the diaphragmatic tears, full lung re-expansion prior to the

closure of the diaphragmatic perforations followed by correction of the intra abdominal injury. Through the laparotomy, all three body cavities were adequately taken care of. I wonder if the believers in thoracotomy for diaphragmatic perforations would have submitted such a patient to bilateral thoracotomy plus a laparotomy. If not, then we are in agreement in principle, at least. It cannot be stressed sufficiently how important it is to follow the technique or the closure of the diaphragm and complete evacuation of the pleural space as discussed in detail in the presentation. Complete evacuation of the pleural space and full lung re expansion must be accomplished in order to avoid subsequent pleural complications.

I wish to repeat that thoracotomy alone, in cases of thoracoabdominal injuries, is dangerous because the survey of the intra abdominal organs cannot be complete through the diaphragm. By this method, cases with missed bowel perforations are known to many of us. The choice of procedure in my estimation, is the one which is the least extensive and accomplishes the most, that is, laparotomy with extension of the wound into the chest only in the combined injuries cannot be handled successfully by laparotomy alone, which is the exception rather than the rule. In other words, I do not recommend thoracotomy for diaphragmatic perforation. If the chest needs to be entered, it is done only as an extension of the abdominal incision. This leads me back to my original statement that perforation of the diaphragm is not an indication for thoracotomy.

It is of great value that Dr Shefts emphasized the very rare need for resection of damaged lung tissue. In this series, this was necessary only as an exception and, even in these few cases, consisted of segmental or subsegmental removal of devitalized tissue hardly attached to the rest of the lung.

Dr Woodruff stressed the value of tracheotomy for thoracic injuries. This is a very helpful procedure in well selected cases namely, the ones with considerable lung damage with a great deal of expectoration of blood and mucus, for the purpose of making the nursing care easier and more effective in keeping the tracheobronchial tree clear. As a matter of fact, the very same patient quoted by Dr Woodruff had a tracheotomy. His death was inevitable as the autopsy findings indicated and as was described in the presentation.

The remarks of Dr Williams are of great interest to me since they substantiate a principle applied in the management of the sucking wounds in this series, namely early and complete evacuation of the pleural space by a catheter through the debrided chest opening and removal of the catheter prior to the closure of the chest wall wound up to skin level. On patients without sucking wounds, in a combat zone tube aspiration is not feasible except when the casualty load is not heavy.

I feel greatly indebted to Drs Berry, Graham, and Samson, who arranged the presentation of this paper in absentia on a few days' notice, and to Dr Wylie for being kind as to accept the burden of reading the paper.

THE REPAIR OF TRACHEAL DEFECTS WITH FASCIA AND TANTALUM MESH AN EXPERIMENTAL STUDY*

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UNTIL recent years surgical treatment of the trachea has been concerned primarily with the sequelae of infections, such as diphtheria, repair after trauma, tracheotomy and, infrequently, with neoplasms. One of the earliest reports concerning a bolder surgical attack on the trachea is that of Burkert in 1918. In this report he summarized experimental and clinical reports of end to end anastomosis, resection of annular segments, and described transplantation of various tissues into surgically created defects of the trachea. Recent interest in the repair of tracheal defects has been concerned principally with techniques, which may be used after removal of neoplasms infiltrating the trachea either primarily or secondarily.

In animals, Taffel and Daniel have reported the repair of small defects created by excision of portions of the cervical trachea and of closure of the end of a main bronchus with autogenous fascia. They concluded that excellent healing took place in such grafts. Fascia has been reinforced by a spirally coiled wire¹ or woven wire strands, steel wire mesh, and tantalum mesh with varying degrees of success.^{2-9, 17} Dermal grafts reinforced with wire have been used.¹⁰ Several types of intubing tube prostheses have been used successfully in the laboratory and clinically in at least one case.^{4, 6, 11, 13} The bifurcation of the trachea of the dog has been successfully resected by several different methods^{6, 7, 9, 12} and, more recently, the feasibility of implantation of a bronchus into tracheal windows has been under investigation.^{15, 16} Homografts of tracheal segments have only rarely given good results, and are ordinarily observed to undergo necrosis with resorption of cartilaginous elements.^{3, 14} Davies and co workers reported that they obtained survival of cartilage homografts when they used an intubing tube support for the graft. The condition of the cartilage after they removed the intubing tubes has not yet been reported.

Our report deals with repairs of the intrathoracic portion of the trachea of dogs and covers the use of autogenous fascia and tantalum mesh, separately and in combination. A few studies were made in which fascia was combined with autogenous cartilage either cut in the form of tracheal rings or ground finely.

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METHODS

Healthy adult mongrel dogs weighing between 10 and 22 kilograms were used. Anesthesia was obtained with ether administered through an endotracheal tube by means of an insufflation apparatus. Exposure of the trachea was effected through an incision in the right fourth intercostal space.

Group 1 Fascia—There were sixteen animals in this group. 'Windows' which included segments of two to four rings and varied in size from 1 by 1 cm to 2 by 2.5 cm were made in the right lateral wall of the trachea of all animals. The fascial patches were obtained from the right paravertebral muscles in most animals, rarely fascia recta was used. The patches were cut large enough so that at least 0.5 cm would overlap the trachea around all margins of the defects. Fascia was applied by means of interrupted sutures in each corner of the patch followed by a continuous suture which went through cartilage rings and fascia. The suture material was 000 chromic catgut. If these patches were applied without too much tension they were airtight.

Microscopic study of sections of the graft and adjacent trachea stained with hematoxylin and eosin was carried out at intervals of two weeks to six months after operation.

Group 2 Fascia and Cartilage—In this group there were eleven animals in which windows were made as previously described. Cut or ground cartilage is applied to the external surface of fascia grafts which were made as described for Group 1. From four animals long narrow strips of costal cartilage were removed with a scalpel. These strips, which tended to curl like a tracheal ring, were fixed to the fascia by means of interrupted catgut sutures carefully placed through the external fibers of the fascia. One or two of these cartilage rings were used in each of the four animals and gave the graft area a convex outer surface.

From the other seven animals cartilage was removed from the tips of lower ribs and ground with a chondrotome. In three animals the fragments of cartilage were applied to the fascial grafts with a piece of Geltoim and in four were spread on a freshly made blood and bovine thrombin* coagulum which had been applied on the external surface of the fascia grafts.

Group 3 Fascia and Tantalum—These materials were used for a total of ten animals in which tracheal defects were made as described for Group 1.

Small Patches—Patches of fascia were attached to one surface of a piece of tantalum mesh by catgut or cotton sutures. The edges of the mesh were carefully folded over so that sharp ends of wire did not protrude. Such patches were made of the same size and shape as the defect. In three animals fish lined the interior of the patch and in four mesh was on the exterior†.

Large Patches—In three animals pieces of tantalum mesh large enough to cover the normal trachea on all edges of the defect were used. After the defect in the trachea had been repaired with a patch of fascia the tantalum mesh was placed over it and the adjoining portion of the trachea, and it-

*L. V. L. thrombin was diluted so that there were 100 units per cubic centimeter.

†Tantalum mesh was obtained from the Ethicon Suture Laboratories; wire was 0.003 in diameter.

tached to the fascia by interrupted catgut sutures. Finally, the tantalum mesh was molded by pressing it against the metal endotracheal tube being used for the anesthesia.

Group 4 Tantalum Mesh With Temporary Prosthesis—In this group there were eight animals. Large windows were removed from the trachea in two animals, and tubular resections with removal of the entire circumference of the trachea were carried out in six. We repaired these defects by placing a plastic tube prosthesis in the lumen of the trachea and covering it with tantalum mesh. Before resection was performed, the plastic tube was introduced into the trachea through a transverse incision. To seal the airway, two loosely twisted ligatures of three strands of No. 1 chromic catgut were tied around the trachea, one close to each end of the tube. Then a portion of wall of trachea or a tubular segment including seven to eleven rings was excised between these ties. The size of the remaining gap, in animals in which tubular segments were removed, was determined by the tension of four interrupted 000 cotton sutures which passed from one end of the trachea to the other over the surface of the tube. This reduced the gap to about 2 cm. A piece of tantalum mesh with folded margins which was large enough to lap over the edges of the trachea at each end was placed snugly around the exposed portion of the tube. The mesh was held in apposition to the tube by means of cotton sutures placed in the mesh which served to tighten the mesh around the trachea.

The intubating prostheses were polyethylene tubes* 6 cm. in length with a wall thickness of $\frac{1}{16}$ inch (0.16 cm.), having two sizes of lumen. One had an internal diameter of one-half inch (1.27 cm.) and another of five eighths inch (1.59 cm.). These tubes were smooth except for three shallow encircling grooves on the exterior near one end which had been made by pressing a heated wire into this surface. The caudal ligature encircling the trachea was tightened around this grooved area to prevent the tube from being dislodged during the postoperative period. The smooth surface and shallow grooving allowed ready removal with bronchoscopic forceps within one to seven months after operation. Periodic bronchoscopic observations were made of the tube and later, after the tube had been removed, of the area of the graft. After the animals were killed, sections made of tissues carefully peeled from the tantalum mesh were studied microscopically.

Group 5 Tantalum Mesh and Fascia With Temporary Prosthesis—These were used on four animals.

Repair of tubular defects was accomplished by a method similar to that used in Group 4 but modified as follows: (1) A sheet of autogenous fascia was wrapped around the defect to cover the exposed tube and the end to end sutures before the tantalum mesh was applied. (2) The fascia and tantalum were fixed to each end of the trachea by a few interrupted cotton sutures, and the tubes were withdrawn two, two and one half, five, and five and one half months after operation.

*Flax Corporation, Hartford, Conn.

RESULTS

Group 1 Fascia—Grossly, all of the grafts looked much alike (Fig 1 *a*). Histologic examination showed that the graft was covered with ciliated epithelium in two instances four to five weeks after operation although one graft was not covered three months after operation. The grafts were flexible and slightly shrunken, a slight to moderate transverse ridge of fibrous tissue covered by epithelium protruded slightly into the lumen. In several dogs the



Fig 1—Segments of the thoracic portion of the tracheas opened to display the grafts. The first pair (a) with fascia and the animal (Group 1) was killed two months after operation. The typical ridge is shown and a tiny ulceration on the center of the graft. *b*—The trachea was repaired with fascia and a strip of cartilage and the animal (Group 2) killed three months after operation. The graft was well healed with practically no deformity. *c*—The trachea with a large fascial and tandem patch (Group 3) was used as specimen obtained two and a half months after operation was well healed and had a good lumen.

normal curvature of the remnants of the cut rings had been lost, and the flattening of the wall opposite the graft which resulted from this produced some further narrowing of the lumen.

The autogenous patch of fascia appeared to have survived in all cases. The evidence for this was that the patches were well vascularized early, there was no decrease in number of cells to suggest gradual death and removal of cells, and in several grafts intact islands of fat cells were noted. The tracheal epithelium seemed to spread across the graft surface with only a minimal granulating base. In fact, epithelium was absent only over a few superficial collections of inflammatory cells or small patches of granulation tissue. In several animals in which the transverse ridge was most prominent and thick, the epithelium on the ridge was nonciliated, cuboidal, or flat. Contracture of the scar appeared to have caused decreased vascularity in the area of the ridge. In two instances tiny, persistent ulcers were found on ridges. There was virtually no regeneration of mucous glands in graft areas, and regeneration of cartilage was confined to islands too small to be apparent on gross examination and located usually near the tips of severed cartilage rings.

Group 2 Fascia and Cartilage—In three of the four dogs in which long narrow strips of autogenous cartilage had been attached to the fascia the mucosal surfaces of the grafts had healed completely when the animals were killed three, four, and five months after operation (Fig 1 b). The other animal had an area of granulation tissue which apparently covered the entire graft two months after operation. Surviving cartilage was present in two of the four grafts, and partial fibrous tissue replacement of the cartilage was noted in one of these two. No grafted cartilage could be found in the other two grafts.

Patches of fascia sloughed partly when ground cartilage had been applied with a piece of Gelfoam in three animals. In the four animals in which a thin layer of thrombin and blood coagulum had been used necrosis of the fascia was minimal. When sloughing had occurred, the grafts lacked epithelium, but in the four animals in which the thrombin and blood coagulum had been used, healing was complete, the areas were firm, and gross bits of cartilage could be seen in examinations at intervals of one, two, three, and four months after operation. Microscopic examination showed that the ground cartilage was incorporated into a fibrous network on the external surface of the fascia, and that there was little evidence of connective tissue replacement of the cartilage.

Group 3 Fascia and Tantalum—Small Patches—None of the three patches applied with tantalum mesh on the inside healed well, and only small portions of the mesh were infiltrated with connective tissue and covered by epithelium. Of the four patches applied with mesh on the exterior, two were completely covered with tracheal epithelium and the other two showed mesh surrounded by granulation tissue within the tracheal lumen. In these latter two animals bronchoscopic examination showed that there had been gradual protrusion of irregular edges of mesh and loose wire ends through the fascia. These animals were killed one to four months after operation.

Large Patches—Weekly bronchoscopic examination of grafts, made of large patches of fascia and tantalum mesh, indicated that these areas were

probably completely covered by epithelium within five to six weeks after operation. No mesh could be seen on any of these examinations, but in one animal a partial breakdown of the surface of the graft was found at the time the animals were killed three and one-half months after operation. In the other two animals the grafts were completely covered by normal ciliated tracheal epithelium which contained no mucous glands (Fig 1, c). Grossly, the graft areas were firm and covered by a fairly dense fibrous tissue which encircled the tantalum. The mediastinal pleura over the tantalum was mobile and normal appearing. Microscopic study showed that there was no inflammatory reaction to the tantalum in any of this group, and there was no evidence of new-formed cartilage.

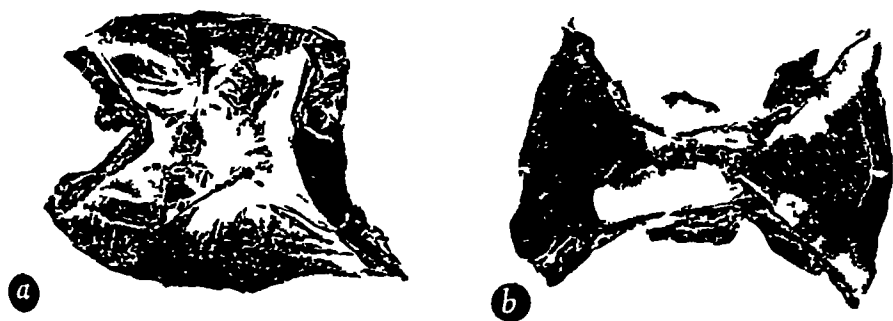


Fig 2—Segments of trachea and graft removed from two dogs in Group 4 in which tantalum mesh was used with a temporary tube prosthesis. *a* Tube was removed four and a half months after operation and the dog died five months later. Tantalum mesh was not much deformed but hourglass stenosis had developed from dense scar tissue on the inner surface of the tantalum mesh. This graft contained several incomplete new-formed cartilage rings. The central split resulted from opening specimen after fixation in formalin. *b* Tube was removed one month after operation and the dog was killed four months later because of tracheal obstruction. The cephalic part of the graft had become detached from the trachea. The deformed tantalum mesh and the fibrous band connecting mesh with trachea are easily

Group 1 Tantalum Mesh With Prosthesis—The polyethylene tubes were fairly well tolerated by all dogs of this group. Although these dogs had short periods of vigorous coughing they had an excellent airway as long as the tubes were in situ. On bronchoscopic examination mucoid secretion could be seen distal to the tubes in a few of the animals but apparently all were able to get rid of this by blowing it out during expiration. Roentgenograms of the thorax which were made at intervals of one to two weeks after operation were consistently clear, and all animals gained weight between operation and the time of death.

Removal of the intubing tubes after periods of from one to seven months was followed in seven of the eight animals by progressively increasing stenosis, and at the time they were killed or died they had respiratory obstruction (Fig 2, a). The length of time the tubes were in situ did not seem to be related to the degree of stenosis; for example, removal of a tube from one dog seven months after operation was followed by almost complete stenosis within one month. Survival after the tubes were removed was as follows: one and one

half and eight months for the two animals which had undergone resection of a small portion of the trachea, and two weeks, one, four, four and one half, and four and one-half months for five animals from which large tubular segments had been removed

Grossly, the graft areas were firm, and the tantalum was enmeshed in connective tissue. The cephalic segment of trachea had retracted from the tantalum mesh in one dog, and a thin ring of fibrous tissue lined by epithelium was found connecting the mesh and the end of trachea (Fig 2, b). The tantalum mesh was grossly deformed in this and in four other specimens, probably the result of contraction of scar tissue. The only animal in this series in which the polythene tube had become dislodged had no deformity of the mesh. We thought this tube had been expelled spontaneously, but it was found that it had shifted down to the bifurcation of the trachea and lodged there where it continued to support the graft. In the seventh animal, the tantalum was still essentially tubular, but a dense layer of fibrous tissue had developed inside the mesh beneath the epithelial lining and produced an hourglass stenosis.

More or less complete covering of these grafts with epithelium was obtained in only three of the seven animals which died or were killed. In two of these three animals, post-mortem changes were marked, and positive identification of epithelium was not possible. The other four grafts were covered by thick beds of granulation tissue. Regeneration of cartilage was found in only two grafts, in one it consisted of several thin, irregular and incomplete rings of cartilage, and in the other there were several small scattered islands of regenerated cartilage.

One dog in which a tubular resection of trachea had been performed had a completely healed graft area with a good lumen when killed sixteen months after operation. When the polythene tube was removed five and one half months after operation, healing of the graft appeared more advanced than in any of the other dogs, and within two weeks the area seemed to be completely covered by epithelium. The graft remained unchanged from this time until the dog was killed ten months later. At post-mortem examination we found some flattening of the mesh in an anteroposterior direction. Tracheal epithelium completely covered the operative defect despite considerable irregularity of the surface (Fig 3, a). The intercartilaginous tissues of two to three segments just cephalad to the graft area were stretched to two to four times normal size and produced a soft conelike narrowing of the lumen (Fig 3, b). This suggests the possibility that the rigid graft may have imparted an excessive amount of strain to the adjacent tissues through the range of tracheal motion associated with breathing and coughing. This was the only specimen in our series showing such a change. The fact that the animal survived longer than any other in the group may or may not add significance to this observation.

Group 5 Tantalum Mesh and Fascia With Temporary Prosthesis—Three of the four animals of this group died or were killed because of tracheal stenosis following removal of the tubes. In one animal the tube was in place for two and one-half months and severe stenosis developed within the next two

months after removal of the tube. In this instance the internal surface of the graft was mainly a bed of granulation tissue beneath which the fascia could be identified grossly and microscopically. There was microscopic evidence of an acute inflammatory reaction throughout the graft which was more marked than usual. In the second animal the tube was in place for two months, and then during the next four months symptoms of obstruction developed despite the bronchoscopic observation of the presence of epithelium. Post-mortem examination revealed a gap between the mesh and the cephalic portion of the trachea, a gap bridged by fibrous tissue which was not rigid enough to prevent collapse during inspiration. Approximately one-third of the graft had a new covering of epithelium surrounding the usual bed of granulation tissue.

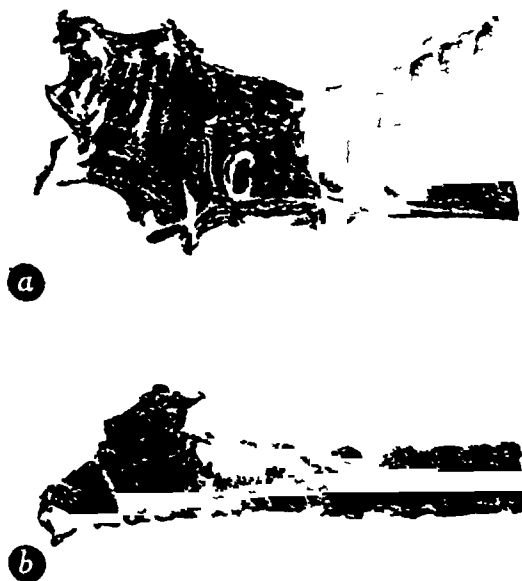


Fig. 3.—a Successful graft in animal of Group 4; graft was covered by epithelium six months after operation and ten months after removal of the tube. b Stretching of inter-laminous tissue cephalad to the graft with soft constrictive narrowing should be noted.

The third dog had the tube removed five and one-half months after operation and died two months later. The tantalum mesh was irregular but had not collapsed and the lumen was decreased to 3 to 4 mm. in diameter by thick stimulation tissue which had not been covered by epithelium.

The fourth dog in this group got along better. This tube was removed five months after operation and the graft seemed rather well healed. Bronchoscopic observation one month later disclosed part of the upper border of the mesh uncovered in the lumen of the trachea. In the next five and a half months until the animal was killed the tracheal lumen decreased only slightly. The more mesh seemed to protrude into the lumen. At post mortem exami-

nation the graft area was flattened in an anteroposterior direction and about a third of the upper margin of the mesh was uncovered. About half of the graft area was covered by epithelium (Fig 4)

COMMENT

The results in our series of repairs with plain fascia are essentially those obtained by others who have described rapid covering of these patches by ciliated tracheal epithelium. Our histologic sections, however, suggest that the autogenous fascia had survived instead of being replaced by new connective tissue and that the epithelium had covered the fascia without need of a granulating base. We were unable to find evidence of regeneration of tracheal mucous glands in the new-formed epithelium. Regeneration of cartilage was observed in a few grafts, a finding reported by others, but it was not sufficient to give adequate tissue support in any instance.



Fig 4—Longest survival in Group 5—tantalum with fascia—shows exposure of mesh and only partial epithelial covering of the graft area.

The survival of some of the cartilage grafts was of interest in view of the relatively avascular bed which must surround and nourish such a graft. Our study suggests that thin strips of cartilage might be useful for support of fascial repairs of defects of limited size in man.

The tantalum mesh used in these studies was not responsible for any specific inflammatory reaction on the part of the host tissues. Frequently, the mesh was incorporated in rather thick, firm fibrous tissue, yet microscopic study showed no evidence of any inflammatory reaction to the metal. Rigidity supplied by the mesh was insufficient to prevent deformity caused by contraction of the fibrous tissue. Protrusion of irregular surfaces of mesh or loose wires appeared to be the cause of failure to develop an epithelial lining and of the erosion seen over such areas. However, there was no evidence that the mesh caused damage to adjacent great vessels and esophagus. From these studies it appears that the development of an adequate lining when tantalum mesh is used in repair depends on the following practices: (1) careful folding of mesh edges to eliminate sharp wire ends, (2) careful molding of mesh to the shape of the trachea, (3) use of pieces large enough to lap over the edges of the trachea for at least 1 cm, and (4) placement of the mesh on the outside of the graft.

Temporary use of an intubating polythene tube to support the area of repair was tested because it promised to obviate some difficulties reported in studies in which wire mesh had been used to reconstruct tracheal defects.^{2,17} With such a tube, airtight reconstruction of the airway may be accomplished easily and rapidly. The previously reported finding⁴ of epithelium in the area surrounding intubating plastic tubes offered the hope that regeneration of epithelium might occur about a removable tube. It is possible that results might have been improved if we had employed tubes of a different design which fitted better and were left in situ longer. It has been found¹³ that polythene tubes of another design prevented stenosis when they were retained for about a year. Under these conditions the fibrous tissue bridging the gap in the trachea became as rigid as the trachea. After removal of the tube, epithelium covered the small, scattered granulating areas, and contraction or significant deformity did not ensue during an observation period of two years.

In the present study we made no attempts to dilate the stenosed part as stenosis was developing.

SUMMARY

Defects of the thoracic portion of the trachea were created in dogs by surgical excision of portions of the wall varying in size from one-half to full circumference and two to eleven tracheal rings in length.

In one group of dogs only autogenous fascia was used to cover small defects. Such grafts survived and were quickly lined by tracheal epithelium. These grafts sometimes contained insignificant amounts of new-formed cartilage but did not possess or acquire sufficient rigidity to warrant their use over large defects.

Autogenous cartilage was placed on the outside of patches of fascia in a group of dogs and was found to survive. Tantalum mesh was used in other dogs after resection of a relatively small segment of trachea to give support to the graft of fascia. The mesh was well tolerated but tended to be extruded into the tracheal lumen when it was not carefully placed outside of the fascia.

In another group of dogs in which a large portion or long tubular segment of the thoracic part of the trachea was excised temporary internal support for the area of the repair was provided. Tantalum mesh or fascia covered by tantalum mesh was applied around intubating polythene tubes. The tubes were left in situ for varying periods before removal. Regeneration of epithelium did not take place with sufficient rapidity in the majority of the animals to prevent tracheal stenosis after removal of the tubes. Use of a sheet of autogenous fascia to line the mesh segment tended to improve the end results.

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CONGENITAL ESOPHAGOBRONCHIAL FISTULA IN AN ADULT

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CONGENITAL fistulas between the esophagus and the respiratory tubes were first noted by Gibson in 1696.¹ However, the variations in this anomaly were not adequately described until 1929 when Vogt² and later Lanman³ classified them. The diagnosis and treatment of these defects were evolved largely through the efforts of Haight and Towsley⁴ who performed the first successful anastomosis in esophageal atresia with tracheoesophageal fistula, and Ladd⁵ who successfully restored esophageal continuity by the construction of an antethoracic esophagus.

Many theories have been advanced to explain the occurrence of these anomalies but their definitive cause is unknown. However, the suggestion that aberrant vessels may play a role in preventing complete separation of the trachea and esophagus seems pertinent in the case we are reporting. Communications between the trachea or bronchi and the esophagus are said to occur about once in every 800 births⁶ but they are rarely seen in adult life unless they are acquired. Monseurat reviewed the world literature on this subject in 1941 and found reports of 222 congenital and 448 acquired tracheoesophageal fistulas. However, this figure probably represents but a small portion of their actual number.

Acquired fistulas may be due to neoplasms, infections (lung abscess, suppurative lymph nodes, empyema, tuberculosis, syphilis, and actinomycosis), trauma, foreign bodies (sharp objects swallowed, broncholithiasis⁶), esophageal diverticula with infection, and they may also arise without demonstrable cause. The etiological factors, symptoms and signs, diagnosis and treatment of these lesions have been admirably summarized in recent articles.⁷

CONGENITAL RESPIRATORY ESOPHAGEAL FISTULAS IN ADULTS

We have found reports of congenital tracheo- or bronchoesophageal fistulas in adults in only four proved cases. One of us¹³ reported the case of a boy 23 months of age with tracheoesophageal fistula complicated by empyema which may have been congenital in origin. Morton and his associates¹⁴ found an esophageal bronchial fistula in a 26-year-old man with associated bronchiectasis which was probably congenital. It was resected and a right lower lobectomy was done with good results. Berglund¹⁵ described a 62-

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year-old man who began having symptoms suggestive of tracheal aspiration at the age of 20 years. The patient was not treated surgically and the diagnosis is based mainly upon x-ray studies. Imperatori¹⁸ observed an 8 year old boy with tracheoesophageal fistula. This child was operated upon shortly after birth for a pyloric obstruction. Later repeated esophageal dilatations were done without relief. Then the fistula was dissected out and tied but it recurred. At a second operation a purse-string suture was placed around the fistula with good results. No biopsies were taken although the fistulous tract was well defined and was probably congenital in origin. Haight¹ reported a well-documented case of a 4-year-old boy with a congenital tracheoesophageal fistula which was diagnosed at birth by Lipiodol studies. The child continued to have a persistent cough and recurrent attacks of pneumonitis. The fistula was successfully divided through a retropleural approach with good results.

Our case is reported for the following reasons: (1) It is rare to find congenital fistulous communications in the adult between the esophagus and the respiratory tubes, (2) it is particularly unusual to encounter such fistulas between the esophagus and the basal bronchus of the left lower lobe at any age, (3) the association of vascular anomalies which may be important in the embryogenesis of such communications as shown by Keith and Spicer¹⁵ and Kraus¹⁹ are illustrated in this patient.

CASE HISTORY

Mrs. R. J. (No. 203306), a Negro woman, 40 years of age, was admitted to the Indianapolis General Hospital on Dec. 6, 1951, complaining of almost continuous productive cough with hemoptysis, chills, fever, loss of weight, anorexia, and extreme weakness. The patient was obviously seriously ill and as we first observed her we noted extreme dyspnea, cyanotic lips, pungently foul breath, frail and emaciated body, and clubbed fingers with advanced osteoarthropathy. We attributed her inability to relate a coherent medical history to the severe illness and also to mental retardation. Therefore, much of our information came from her husband and relatives.

The symptoms had begun in very early childhood, in fact, she could not remember when she had not coughed and brought up sputum. Members of her family informed us that she was a "sickly baby" and that she had a persistent cough at birth and "strangled easily" when she was fed. Throughout her entire life she had been in and out of hospitals. A few of the more significant episodes in her long medical history should be enumerated. The first twenty-five years of her life were marred by constant illness. Although she had been in the hospitals of many cities in the South we were unable to get accurate descriptions from the family or from the patient of the complaints or diagnoses or even the names of any of the hospitals. We do have a record of her admission to a hospital in Nashville, Tenn., in 1937 after a severe attack of pneumonia. She stated that such attacks were very common but that this time the "pneumonia" persisted and the doctors had to remove two ribs, pus drained from the chest for many months. Following her release from the hospital in Nashville, she traveled to many cities and had repeated attacks of what he called pneumonia with a persistent cough, constant tiredness, frequent episodes of hemoptysis, and failure to gain weight. These symptoms led to a diagnosis of tuberculosis.

She was first seen in the Indianapolis General Hospital on Dec. 31, 1941. At this time a diagnosis of multiple abscess of the left lower lobe with severe bronchiectasis was made. The sputum and gastric washings were examined for acid fast bacilli repeatedly but none were found or cultured. The predominant organisms found were chiefly streptococci, staphylococci, and fusiform bacilli. She was referred to the Chest Service at this

time and a lobectomy was advised. However, after a severe coughing episode an examination of the right upper lobe in addition to the disease in the left lower lobe. The erythrocytes at this time numbered two million and the hemoglobin 6 Gm. A progress note on the chart states that the patient's condition was too precarious to permit surgery. During the next three years the patient came to the Indianapolis General Hospital on the following dates: June 15 to July 15, 1943; Nov 8, 1943, to Jan 5, 1944; Oct 5 to Oct 13, 1944; May 2 to May 14, 1946; June 9 to Aug 24, 1946; Nov 10 to Dec 1, 1946. Her chief complaints on every admission were productive cough (10 to 12 ounces of pus daily), hemoptysis, chills and fever, loss of weight, and weakness. The findings on x-ray examination, bronchoscopy, and bronchography indicated a progressive sacular bronchiectasis with multiple abscess of left lower lobe. Lobectomy was advised each time but refused. Treatment consisted of multiple blood transfusions, postural drainage, antibiotics, and chemotherapy.



Fig. 1—1. Right bronchogram showing normal bronchial tree. Note the Lipiodol which remains in the left lower lobe from bronchography which had been done six weeks previously. 2. There is no filling of the Lipiodol in large bronchiectatic cavities two weeks after the left bronchogram had been repeated.

During the next five years the patient entered many hospitals in other cities and finally returned to the Indianapolis General Hospital on Dec 6, 1951, when we saw her for the first time.

One day prior to admission she coughed up approximately 5 to 12 ounces of blood. This frightened the family and she was brought to the hospital prepared to accept the advice of the hospital staff. A complete work up was started and it was found that many of the points in her past history had not been recorded on previous admissions. She had never been able to play with other children because of extreme shortness of breath. At the during coughing periods she would cough up small particles of food milk, and cake. After the thoracotomy in 1952 the food particles which she coughed up were much larger and more frequently observed. Physical examination at this time showed a flat in the lower left chest posteriorly. The entire left lung was dull. There was a 1 cm. murmur heard best over the fourth inter space to the left. Bronchoscopic ex-

amination showed a stenosis of the left lower lobe bronchus, and bronchography revealed multiple abscesses in the left lower lobe and advanced bronchiectasis (see Fig 1) Some of the Lipiodol which had been injected into the bronchial tree was present in the stomach and for this reason it was decided that a barium swallow should be given. Fluoroscopic observation and x ray studies showed that some of the barium found its way very easily into the bronchial tree on the left side (see Fig 2). It was difficult to give the patient

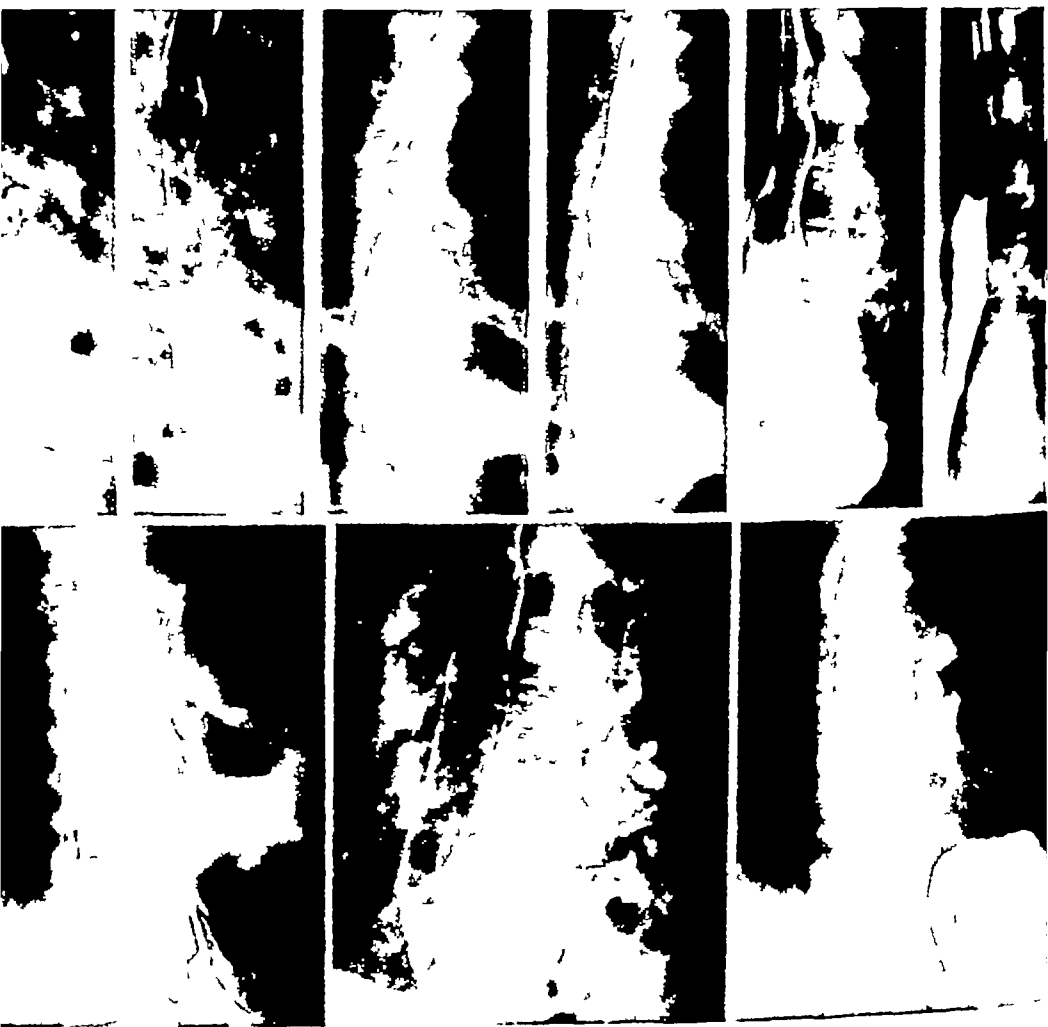


Fig 2—Barium swallow. X-ray film taken at one minute intervals. Note that the left lower bronchial tree is well filled within eight minutes after the ingestion of barium. It is highly probable that the opaque material was propelled by contraction of the smooth muscle in the fistulous tract.

a barium meal because of severe vomiting and therefore the diagnosis of cardiospasm or achalasia were considered. The pulmonary disease could thus be due to aspiration rather than to direct communication. However, after repeated attempts enough barium was retained to demonstrate a bronchoesophageal fistula. The vital capacity was approximately 30 per cent. Erythrocytes numbered 2,900,000, hemoglobin 9 Gm, and leukocytes 14,000. In

pite of the precarious condition of the patient we decided that operation should be done and therefore she was prepared with antibiotics and multiple blood transfusions and exploratory thoracotomy was performed on March 1, 1952.

At operation the visceral and parietal pleurae were found to be densely adherent so that access could not be gained into the pleural cavity without tearing the friable lung and therefore the lung was mobilized by an extrapleural approach. The esophagus was identified and a fistulous tract was encountered which communicated between the esophagus and the basal bronchus of the left lower lobe. The fistula was well defined and was a tubular structure approximately $1\frac{1}{2}$ cm long and about 6 mm in diameter (see

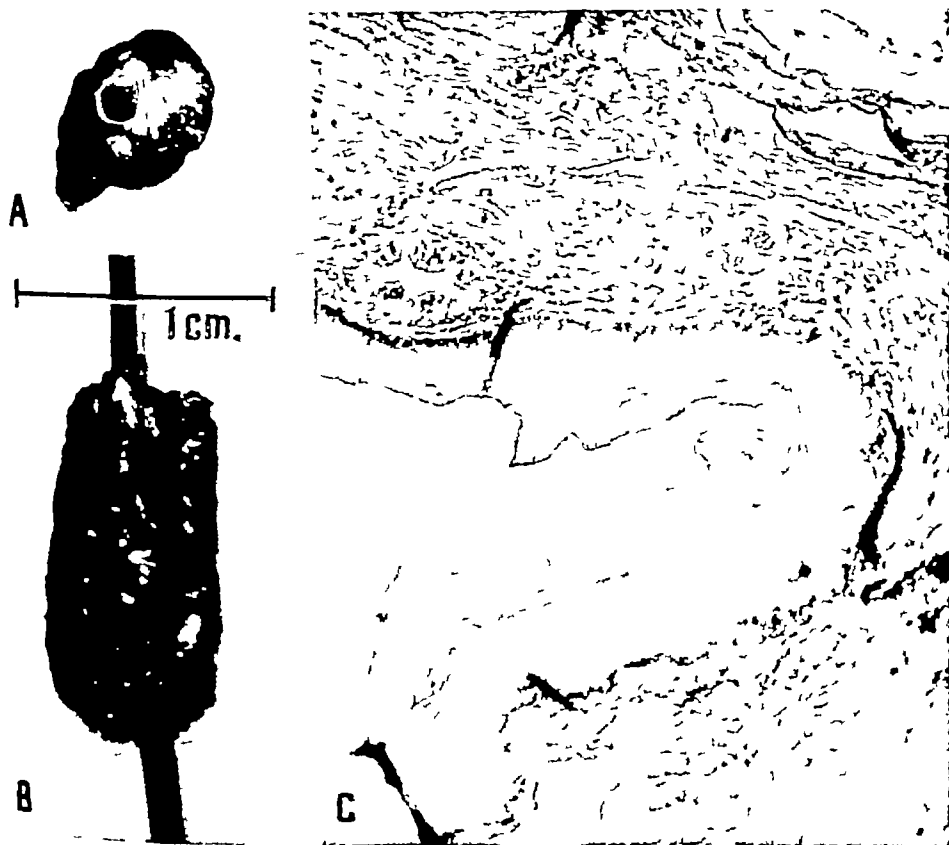


Fig. 2.—A: Photograph of the excised esophagobronchial fistula showing the diameter of 6 mm. B: The fistula has been threaded over a small catheter to show its length. C: Photomicrograph showing the various layers normally found in the esophagus together with glandular elements. (See text for full description.)

Fig. 3) The fistulous tract was dissected out and removed close to the esophagus. The opening in the esophagus was closed with interrupted 0000 silk. A cuff of fistula was left on the bronchial side which was closed by interrupted 0000 silk sutures. Although a left lobectomy and lingulectomy was contemplated it could not be done because of a severe and alarming fall in blood pressure in spite of ample blood replacement. Finally the chest was closed in layers using 000 silk throughout. The postoperative course was marked gratifying and surprising. Symptoms abated immediately, there was no coughing, no recurrence of hemoptysis, and the patient stated that she felt better.

Microscopic examination* was as follows. Sections through this tubular specimen showed a lining of normal stratified squamous epithelium. The underlying tissue was dense connective tissue which was rather heavily infiltrated with lymphocytes and plasma cells. Outside of this, there was a fairly thick layer of smooth muscle. Within the connective tissue beneath the epithelium several mucus secreting glandular structures were seen similar to those found normally in the esophagus. No other unusual features were noted in this specimen. Diagnosis was esophagobronchial fistula, congenital.

Eighteen days later the old incision was reopened and the lung freed by extrapleural dissection. This was the only feasible course to pursue since it was impossible to gain entrance into the pleural cavity because of the extremely dense adhesions which existed between the visceral and parietal pleurae. Upon approaching the pulmonary artery it was found that a patent ductus arteriosus was present. This was clamped and divided and closed with a continuous everting mattress suture of 00000 silk supplemented with an over and over continuous stitch of the same material. The pulmonary artery side was removed together with the pulmonary artery after ligation proximal to this point. As the individual hilar ligation proceeded it was noted that a second artery was present arising from the aorta immediately above and adjacent to the site of the esophagobronchial fistula and communicating with the basal branch of the pulmonary artery. It was doubly ligated and divided. The lung was removed and the bronchus sutured with interrupted 00 silk sutures. The chest was then closed in routine manner. The patient's post operative course was entirely uneventful and she was released from the hospital on March 31, 1952, twelve days after pneumonectomy.

The pathology report was as follows:

A. The left lung showed a fairly normal appearing upper lobe. The lower lobe was roughened and very firm and showed numerous dense adhesions over its pleural surface. The adhesions extended upward along the posterior surface of the upper lobe. Examination of the hilar region showed in one area the presence of a black silk suture about a small piece of fibrous appearing tissue entering into the pulmonary artery which was stated to be a patent ductus arteriosus. This small strand of tissue was sectioned and labeled "A" in order to identify it. Another section was taken through the pulmonary artery at its point of entry to identify this junction. This structure was not identified grossly with certainty.

B. Another black silk suture in the hilar region was stated to be a point of entry of a congenital fistula. This area was sectioned and labeled "B" for possible identification. No definite structure was identified grossly. This was in the area of the bronchus leading to the lower lobe.

C. Examination of the upper lobe failed to show any gross abnormalities.

D. Multiple sections through the lower lobe showed the entire parenchyma to be honeycombed with multiple abscesses. These varied from 3 to 3.5 cm in diameter and were all thick walled and surrounded by very dense fibrous tissue. The intervening parenchyma was more firm than normal and appeared to be airless (Fig 4).

Microscopic—Section of the abnormal vessel entering the pulmonary artery disclosed smooth muscle and adjacent bronchial elements. In the section taken from the area adjacent to the previously surgically removed esophagobronchial fistula, black silk sutures were seen imbedded in fibrous tissue, the latter being surfaced on one side by stratified squamous epithelium similar to that found lining the fistula (surgical path No. 527-52 S). Section of the upper lobe disclosed acute congestion of the parenchyma and scanty chronic inflammatory exudate about the walls of small bronchi. The cavities described in the lower lobe, arranged along the continuity of the bronchi, were all lined with respiratory epithelium with the exception of a few areas of squamous metaplasia. These successive dilations of all the bronchi in the lower lobe represented an advanced

* Described by pathologists William L. Drake, Jr. and Paul B. Evans.

degree of saccular bronchiectasis. A dense mantle of chronic inflammatory exudate underlay the bronchial mucosa lining these cavities, and there was atrophy of the muscle and cartilage elements in many places. The parenchyma surrounding these cavities was almost completely fibrotic with numerous foci of chronic inflammatory exudate densely thickened alveolar walls lined by "lining membrane" filled with macrophages. Evidence of peptic granuloma was lacking. At several points in the dense, fibrotic parenchyma, there were pherial apertures in the fibrous tissue with 'signet ring' arrangement, the nucleus being off to one side, and the picture suggesting a healed lipid pneumonia.

Diagnosis—(1) Patent ductus arteriosus of pulmonary artery on the left. (2) Postoperative state, recent, following excision of esophagobronchial fistula, congenital, of lower lobe bronchus or left lung. (3) Chronic inflammation, nonspecific and fibrosis, of left lung. (4) Bronchiectasis, saccular, advanced, of lower lobe bronchi or left lung.

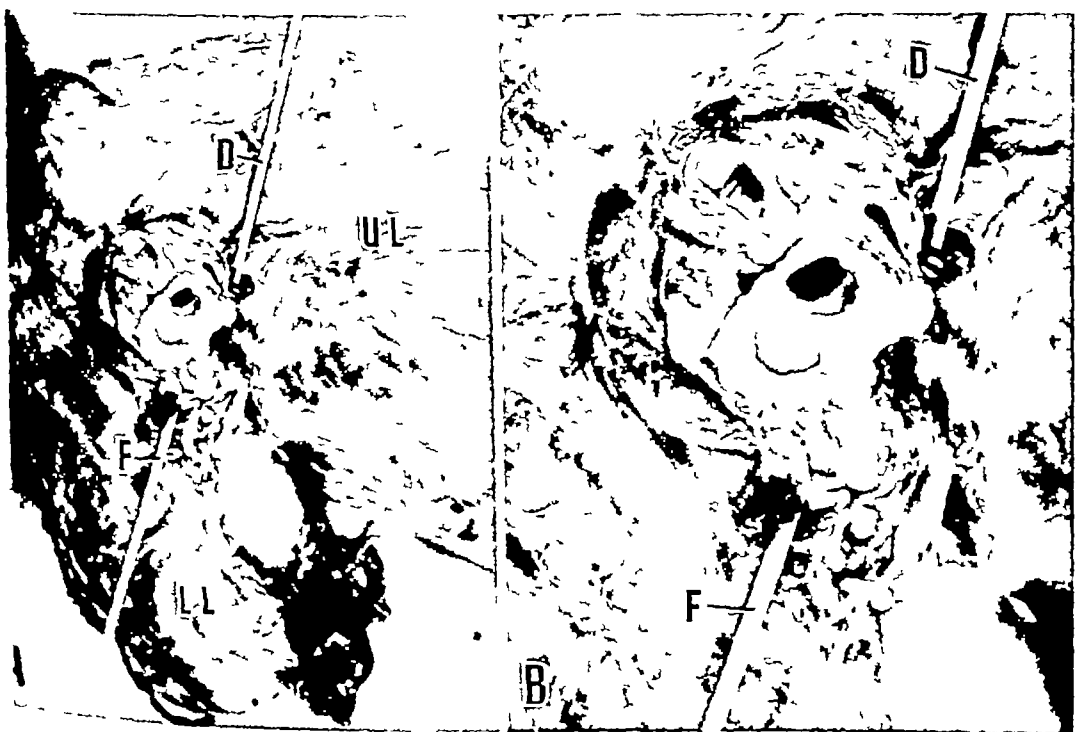


FIG. 1.—A Photograph of excised lung showing the ductus arteriosus (D) and the esophagobronchial fistula (F). Upper lobe (UL) appears to be fairly normal but was found to be somewhat involved after microscopic examination. The lower lobe (LL) is a state and filled with large bronchiectatic cavities. The second ductus is immediately below esophagobronchial fistula (F) but is not clearly shown in the photograph. B Close-up photograph. (See text for full description.)

DISCUSSION

Perhaps many of the so called spontaneous fistulas which occur in adults are really congenital in origin of the type 3C as described by Vogt or the type 3D as described by one of us.¹ Symptoms may be delayed because of a thin membrane which persists for a time or is a result of a valvelike fold venting pulmonary aspiration.

All cases reported in adults gave a long history of persistent cough with recurrent attacks of pneumonitis or pneumonia. This is but another illus-

tiation of the necessity for a careful examination in every patient with persistent cough. Esophagography should be included not only because of possible fistulas but also to rule out a pharyngoesophageal diverticulum, achalasia, cardiospasm, and other defects which might lead to regurgitation and aspiration.

Congenital anomalies are likely to be multiple regardless of where the predominant lesion occurs. However, the association of vascular malformations has not been emphasized as a pertinent factor in the embryogenesis of tracheoesophageal communications except by Keith and Spicer and Kraus. Our patient shows the possible causal relationship between such vascular aberrations and the abnormal communication which was present. The anomalous artery or "second ductus" which connected the aorta to the basal branch of the pulmonary artery was adjacent to the fistulous tract and may have interfered with the complete separation of the esophagus from the trachea which normally occurs at about the fifteenth day of fetal life. The patent ductus arteriosus accounted for the to-and-fro murmur which could be heard and also for the retarded mental and physical development of the patient, although chronic pulmonary disease may have similar effects. Perhaps the two "ducts" may have resulted from a double sixth aortic arch on the left, however, we have found no reports of such embryologic deviations.

CONCLUSIONS

1 A case of probable congenital fistula between the esophagus and the basal bronchus in a 40-year-old woman is reported.

2 Associated vascular anomalies were discovered. These consisted of a patent ductus arteriosus at the usual site and an aberrant artery leading from the descending aorta to the basal branch of the left pulmonary artery.

3 There was immediate improvement following ligation and removal of the fistulous tract, and complete recovery following extrapleural left pneumonectomy with ligation and excision of the two ducts.

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CALCIFIC EMBOLI COMPLICATING MITRAL VALVE COMMISSUROTOMY

REPORT OF A CASE

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INTRODUCTION

THE increased frequency with which mitral valve commissurotomy is being performed makes it imperative that all its possible complications be recognized so that they may be averted, if possible. Since the operation deals with a valve which is frequently heavily calcified, it was early recognized that the manipulation might dislodge some of this calcific material. Fear of this complication was strengthened by the death of several patients who presented the signs and symptoms of cerebral infarction immediately following surgery. In these patients there were no mural thrombi in the left auricle or atrium. Autopsy failed to recover any calcific emboli in these cases.

We are presenting a case in which the calcific emboli, which originated from the manipulated calcific valve, were recovered at autopsy.

CASE HISTORY

A 26 year old white man entered Hahnemann Hospital with the chief complaints of shortness of breath and hemoptysis. During the previous three years he had become aware of gradually increasing dyspnea upon routine exertion. At the time of admission he was dyspneic upon ascending a flight of stairs. During the past year he had three episodes of hemoptysis during paroxysms of coughing. The past history was entirely negative, no knowledge of rheumatic affliction having been known until the diagnosis of mitral stenosis was made two months before hospital admission. An allergic history of repeated, rather severe episodes of ivy poison occurring between the ages of 8 and 12 years was obtained.

Physical examination revealed a normally developed, husky, and well nourished young man with significant findings referable only to the cardiovascular system. There was a diffuse precordial pulsation visible without physical evidence of left ventricular enlargement. Sinus mechanism was present with a blood pressure of 150/70 semirecumbent. In the mitral area, there was a sharp first sound with a grade II, high pitched, blowing, systolic murmur. Left lateral positioning augmented a long, low pitched, rumbling diastolic murmur having slight presystolic accentuation. In the third left interspace and along the left sternal border to the apex, there was a grade II, early, high pitched, *decrescendo*, diastolic murmur. P₂ was accentuated two plus and there was simple splitting of the second sound in the pulmonic area and along the left sternal border. A Corrigan type pulse was palpated. No signs of congestive failure were found.

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Electrocardiographic tracings indicated right ventricular hypertrophy with atrial dysfunction. Radiologic studies indicated three plus enlargement of the left atrium, two plus enlargement both of the inflow and outflow tracts of the right ventricle and two plus enlargement of the left ventricle. There was presumptive evidence of calcification of the mitral valve.

Other laboratory studies were within normal limits except for a coagulation time of 15 minutes, 30 seconds. The bleeding time was 2 minutes, 15 seconds. Westergren erythrocyte sedimentation readings were 3 mm per hour.

Because of a discernible degree of aortic insufficiency, with some left ventricular enlargement, assessment of operability was a most problematic task. In view of the severity of the symptoms attributable to pulmonary hypertension associated with moderately advanced mitral stenosis, and realizing the inability to alter the aortic lesion, operation was felt feasible, justifiable, and rational.²

Mitral valvular commissurotomy was performed. The valve was found to be calcified, with some of the calcification having the 'cake icing' quality. Technically the operative procedure was satisfactory.

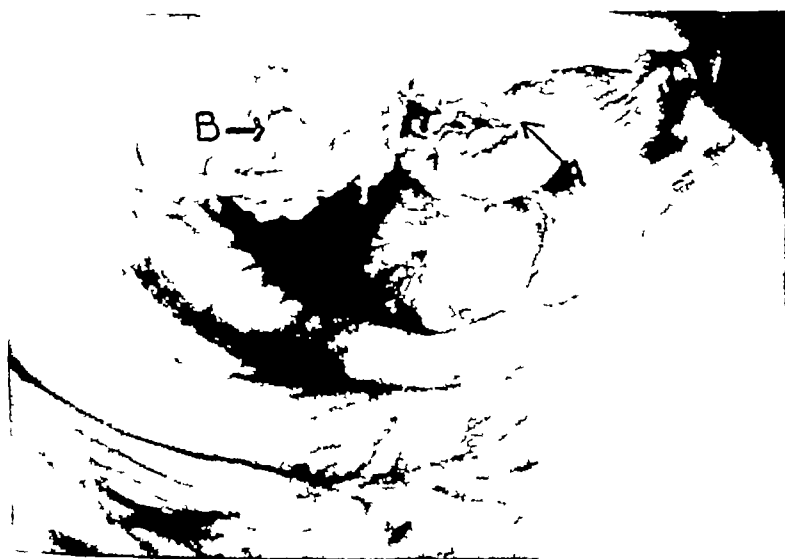


Fig. 1—Atrial view of mitral valve. Arrow B points to granular surfaces of incised lateral commissure. Arrow P points to calcified vegetations on atrial surface of mitral valve.

Postoperatively, the patient had right hemiplegia at the time of partial recovery and the left. He responded sufficiently to recognize acquaintances but no more. A left bundle branch block was performed twice with no appreciable clinical improvement.

Intubation was accomplished. The patient's blood pressure ranged around 110/50 mm Hg twenty hours postoperatively when it became unstable and even imperceptible on the left arm. At that time he developed paroxysmal muscular twitchings with subsequent rigidity of the lower extremities. Tracheotomy was performed because of accumulation of secretions in the tracheobronchial system and frequent aspirations were necessary. The hours prior to death he vomited a moderate amount of dark brown liquid. At that time presented no remarkable findings. Blood pressure readings for the last hour prior to death gradually went from 110/50 mm Hg to 100/50 mm Hg. Temperature suddenly ranging from 101 to 104 F (orally). A pulse rate of 100 min. The respirations at no time exceeded 24/min. The patient died thirty hours postoperatively.

PATHOLOGICAL OBSERVATIONS

Gross Findings—General Autopsy disclosed a left thoracotomy incision which was closed with wire sutures. Also, there was a suprasternal tracheotomy incision.

In the peritoneal cavity there were 200 c c of brown nonviscous fluid located in the left subdiaphragmatic space, and 50 c c of similar fluid within the pelvis. This liquid had the odor of gastric juice. The capsules of the left lobe of the liver and spleen, and the serosal coat of the splenic flexure of the colon, had finely granular surfaces which were friable. Splenic flexure mesenteric tissues were digested, yielding a dissection of the mesenteric vessels. These were intact and stood out distinctly. The serosa of the descending colon and sigmoid colon was covered with numerous ecchymotic areas. An opening was found along the greater curvature of the fundal portion of the stomach which was 14 cm long and extended distally from the esophagus. The edges of this opening were irregular in outline and thin. The mucosa of the stomach contained but few rugae, and most of the lining was smooth, thin, and friable, suggesting autolysis.



Fig. 2—Ventricular view of mitral valve. Arrow points to granular surfaces of the incised lateral commissure.

In the left leaf of the diaphragm there was a hole located in its posterior aspect about 2 cm to the left of the esophageal hiatus. The edges of this opening were irregular in outline, friable, and thin. Four hundred eighty cubic centimeters of fluid were in the left hemithorax, and in character was similar to that found in the abdomen. Pleural surfaces of the left lung were covered with petechiae.

Pericardium In the left lateral aspect of the pericardium there was a recent surgical incision whose edges were approximated properly with sutures. The parietal and visceral pericardial surfaces were covered with a fine fibrinous exudate.

Heart The heart itself weighed 510 grams. On the tricuspid valve leaflets, along the line of closure and on the atrial side, there were firmly attached white verrucae averaging about 3 to 4 mm in size. The chordae tendineae were thin and delicate. Examination of the mitral valve leaflets showed them to be very thick, rigid and shelflike due to calcification. Fusion along both commissures was evident. On the atrial side there were several firmly attached calcified vegetations about 4 to 5 mm in size. The valve must have been stenotic and incompetent before operation. The anterolateral commissure had been incised surgically, and the valve opening now admitted two fingers. The edges of the incised portion of the commissure contained friable calcified material which was dislodged easily by running the finger tip over their surfaces. The aortic valve cusps were slightly

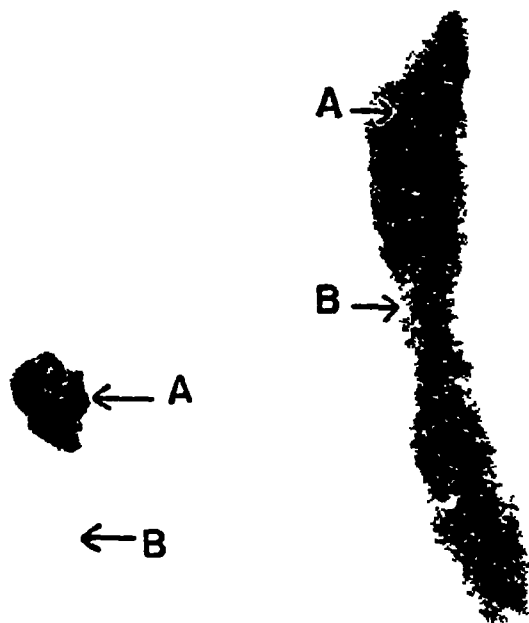


FIG. 3

FIG. 4

FIG. 3—Roentgenogram of the left middle cerebral artery (B) showing radiopaque embolus (A) at its first bifurcation ($\times 3$)

FIG. 4—Roentgenogram of the left posterior cerebral artery (B) showing radiopaque embolus (A) in its lumen ($\times 9$)

flexed but were pliable, approximated one another easily and held a column of water in the aorta without leakage. No fusion was noted along the commissure, and the valve opening admitted two fingers. There was a purse-string suture on the stump of the left atrium. The myocardium was homogeneous. Hypertrophy of the right ventricle produced a maximum measuring 6 mm. The left ventricle measured 15 mm. The right atrium, right ventricle and left atrium were dilated. Inspection of the coronaries proved them to be

Brain Weighing 1,520 grams the brain showed sulci which were narrowed with moderate dilatation, indicating cerebral edema. The meninges were not remarkable, free of thrombi and there were no areas of pigmentation.

At the first major bifurcation of the left middle and posterior cerebral arteries the vessels appeared distended. Palpation revealed stony hard masses at these locations, and these were too hard to cut with a knife. Roentgenographic examination indicated these masses were radiopaque and suggested the presence of calcium. Sections of the left cerebral hemisphere revealed two areas of softening, one corresponding to the area supplied by the middle cerebral artery which involved the medial aspect of the temporal lobe and the entire length of the internal capsule and the nuclei on either side. The other area was in the occipital lobe and seemed to involve all of it.

Kidneys The right kidney contained reddened areas which extended into the cortex.



Fig. 5—Embolus in the lumen of the left middle cerebral artery. Arrow points to calcium particles. Hematoxylin and eosin stain ($\times 28$).

HISTOPATHOLOGY

Microscopic sections of the tricuspid valve leaflets showed evidence of rheumatic activity. Areas of connective tissue edema and fibrinoid degeneration were apparent. In some fields, there were Aschoff bodies on the valve surfaces. Other fields showed organizing verrucae filled with proliferating fibroblasts. In the sections of the myocardium taken from the base of the left ventricle and septum, there were numerous myocardial and perivascular Aschoff bodies. Several of the smaller coronary arteries showed marked rheumatic arteritis, their lumina being occluded by thrombi. These thrombi did not give a positive reaction for calcium with the Kossa calcium stain. No areas of myocardial infarction were seen. The pericardium was covered with a thin fibrinous exudate containing a few acute inflammatory cells.

The sections of the left middle cerebral artery showed a normal vessel wall, but the lumen was filled with a thrombus containing numerous homogeneous basophilic particles which stained black with Kossa's calcium stain, confirming the suspicion that these were particles of calcium. Sections of the left internal capsule and occipital lobe showed areas of infarction with edema, perivascular and intracerebral hemorrhage. Study of sections of the right kidney disclosed two arteries at the hilum which were filled with thrombi.

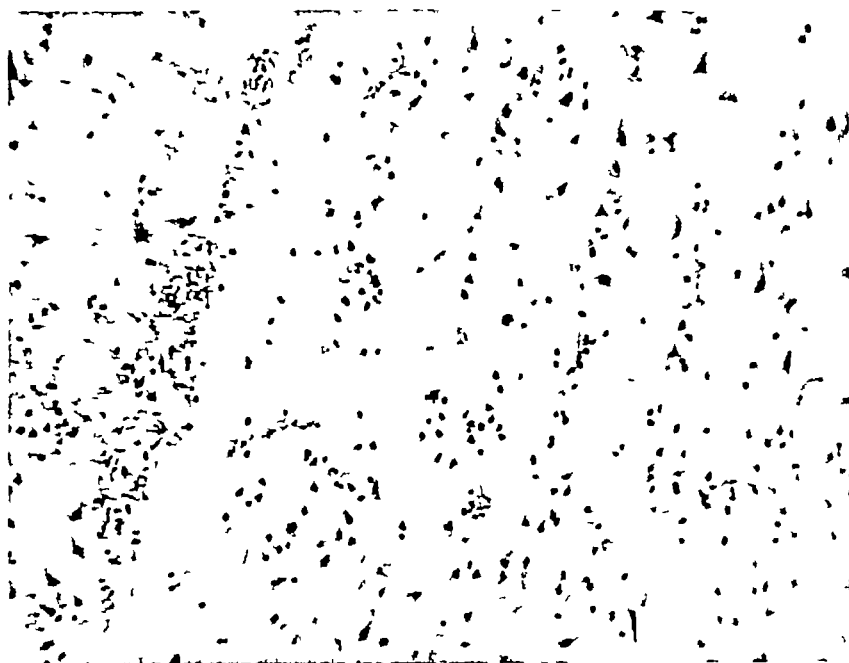


Fig. 2—Early infarct of the left cerebral hemisphere. Hematoxylin and eosin stain. ($\times 96$)



Fig. 3—Thrombus in the right atrium (arrow). Hematoxylin and eosin stain. ($\times 96$)

containing homogeneous basophilic particles which stained black with Kóssa's calcium stain. There were areas of fresh infarctions in the medullary and cortical portions adjacent to these arteries.

The tissue taken from the perforated portion of the stomach and diaphragm showed autolysis of the entire thickness of these structures with no inflammatory reaction.

DISCUSSION

The sequence of events in this case seems to have been as follows. With manipulation of the calcified valve, there was dislodgment of several particles of calcium which embolized the left middle and posterior cerebral arteries as well as the right kidney. The emboli to the brain produced infarction of the areas supplied by these occluded vessels, which in turn resulted in the right hemiplegia and hyperpyrexia.

Lack of reaction around the perforated sites of the stomach and diaphragm indicates that these occurred post mortem. The hyperpyrexia this patient had must have facilitated the development of these digestive changes in the four hours elapsing between death and performance of autopsy.

The threat of this complication is definitely greater when the calcified valve is fractured digitally rather than cut, for then there is less control over the operative site, and calcium particles are more likely to be dislodged if in the way of the probing finger.

No previously reported case of calcium embolism complicating mitral valvular surgery could be found in the literature.

In 1950, Moragues, Bawell, and Shriader¹ reported a case of calcific emboli in the coronary artery system. The origin of these emboli was from a nodular calcified aortic valve without bacterial endocarditis. These had become dislodged spontaneously. All other reported instances of emboli to the coronary artery system concerned valves involved by bacterial endocarditis.

With the hope of obviating future difficulties of this type, Dr. Charles P. Bailey now has developed a refinement of the operative technique, which consists of occlusion of the innominate artery and the left common carotid artery at the critical moment when calcium or thrombi may be expected to be dislodged during manipulation of the calcific mitral valve. This procedure is being published in detail elsewhere.²

SUMMARY

We have presented a detailed study of a case manifesting a very unusual course of events following mitral valvular commissurotomy for the amelioration of mitral stenosis. A calcific embolus was dislodged from the mitral valve during surgery, resulting in occlusion of the middle and posterior cerebral arteries at their bifurcation. Hemiplegia followed with subsequent death within several hours. Calcium emboli were also located in the right kidney.

Calcium from the mitral valve has been suspected as the source of the cerebral vascular accidents following mitral valvular surgery, but this is the first instance in which the embolus actually has been retrieved from the cerebral vessels.

We wish to express our appreciation to Dr John E. Gregory, Dr Charles P. Bailey, and Dr Lowell L. Lane for their interest, advice, and criticism. We would also like to thank Dr Harry D. Lickens for taking the roentgenograms of the cerebral arteries.

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RESECTION IN PULMONARY TUBERCULOSIS

ANALYSIS OF ONE HUNDRED SEVEN OPERATIONS ON ONE HUNDRED ONE PATIENTS, WITH EARLY RESULTS

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SINCE Freedlander¹ performed the first successful planned lobectomy for pulmonary tuberculosis in 1934, resection in that disease has become increasingly safe. Reference to Table I, which summarizes some of the results collected from the literature, will confirm that impression. Undoubtedly, the adoption of the individual ligation technique in the early forties was a great step forward. Numerous other advances, especially in the fields of anesthesia and blood replacement, have made their contributions. However, it was not until 1947, when streptomycin had gained general usage, that early morbidity and mortality figures showed their greatest improvement. Although it is too soon to evaluate the late results of resection performed under streptomycin, reports such as those by Himmelstein, Berry, and Read² are very encouraging.

It is the purpose of this paper to present an analysis of 107 consecutive resections performed on 101 patients at the Connecticut State Tuberculosis Sanatorium, Norwich, Conn., between October, 1948, and July, 1951. Six patients had bilateral resections prior to July, 1951. An additional 3 patients included in this series had contralateral resection without complication subsequent to July, 1951. The latter operations are not included in this analysis. Postresection thoracoplasty was performed in 53 instances without complication. There were 40 men and 61 women. Ages ranged from 15 to 75 years. Ninety per cent of the patients were between 30 and 50 years of age.

A careful study of Fig. 1 will show that our approach to the problem of treating pulmonary tuberculosis by resection was a cautious one. However, it was inevitable that the first patients to have this treatment would be desperate risks. For this reason, a high percentage of the early resections were pneumonectomies. In fact, for the entire series of 101 patients, pneumonectomy was performed in 36, or 35.8 per cent. Another significant observation is that thoracoplasty failure was a major indication for resection in the series. Twenty-eight patients were subjected to some form of resection after having had an unsuccessful thoracoplasty. For comparison, a graph has been made of the number of thoracoplasties performed during the thirty-three month interval of this study. This, of course, does not include the number of postresection thoracoplasties. As the number of resections increased, the thoracoplasties diminished rather abruptly during the last few months. There were 34 pure lobectomies but, in addition, there were 11 lobectomies performed in conjunction with seg-

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TABLE I

AUTHORS	YEARS	ILL CLINICAL STATUS	PER CENT BRONCHO- PULMONARY AND/OR EMPHYSEMA	PER CENT OF FRACTURE MORTALITY	PER CENT FATAL MORTALITY	PER CENT OF TOTAL NO. OF PATIENTS WITH NEGATIVE SPUTUM AT TIME OF LAST REPORT
Thornton and Adams Sweet ¹³ Overholt et al. ¹⁰	Prior to 1912	18.1	37.1	21.7	10.5	
	1910-1915	7.9	9.5	11.3	10.2	33.3
	1911-1912	10.7	11.1	27.7	22.2	13.8
	1912-1911	17.6	12.2	11.8	13.5	51.0
	1911-1910	8.7	7.7	10.6	1.5	
Hammelston et al. ⁷	1917-1950	Tuberculous complications	5.7	% total mortality	5.3	
	1910-1916			27.3	13.6	59.1
	1916-1918			9.8	2.0	72.0
	1910-1911	9.1	18.8	9.1	9.1	
	1911-1916	15.0	27.0	27.0	10.0	55.0
Baker et al. ⁹	1910-1918	1.0	7.0	16.0	0.0	77.0
	1912-1911	6.3	12.5	6.3		75.0
	1911-1917	20.0	25.7	←←←←← 25.6	←←←←← 5.7	81.1
	1917-1919	1.5	2.1	←←←←←	←←←←←	(for selected portion of total patients operated upon)
						75.0 (1950)
Gale et al. ¹²	1915-1918	10.0	2.5	2.5	0.1 (1950)	
	1918-1919		7.5	1.2		
	1915-1919	1.0	11.0	6.9	5.1	71.3
	1916-1950	2.7	8.0	1.0		50.7
	1916-1918	.5	17.5	←←←←← 20.0	←←←←←	75.5
Chamberlain and Klopstock ¹¹ Davieson Johnsson and Ullmar ¹⁷	1915-1950	11.0 (7 of 51)	11.1 (6)	13.0 (7)	3.7 (2)	66.6 (30 of 51)

* Figures adjusted to make them as comparable as possible from available information

mental or wedge resections. The remaining 26 patients had either segmental or wedge resections. These are not shown as such on the chart but are grouped with the "combinations."

Indications for resection, at first limited to thoracoplasty failure, broncho stenosis, tuberculous bronchiectasis, tuberculoma, and lower lobe cavity, were gradually extended to include all of those listed in Table II.

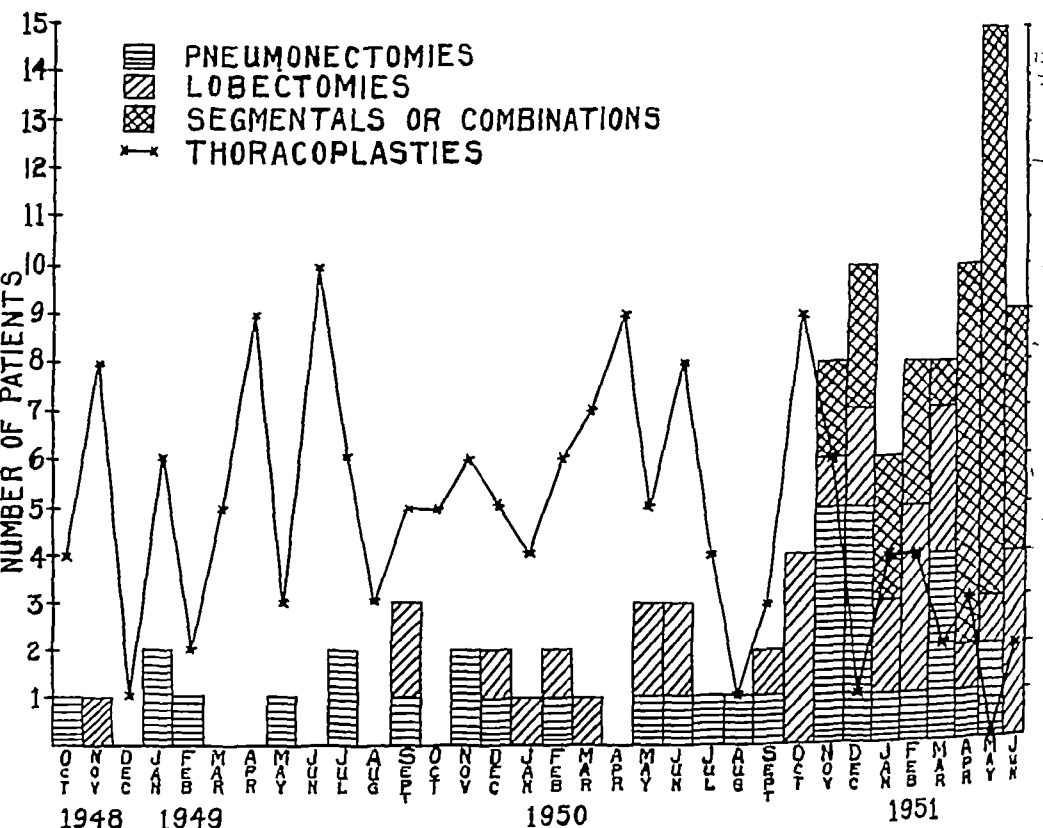


Fig 1

Prior to surgery, each patient was carefully evaluated by the combined medical and surgical staffs. Many patients had received prolonged treatment in the form of bed rest, streptomycin, phenelisis, pneumothorax, pneumoperitoneum, and thoracoplasty. Resection was not performed until lesions in the portion of lung to be conserved were stable, or at least regressive. No patient was refused operation who might benefit from the procedure. In several instances of bilateral, far-advanced disease with cavitation, the cavitory diseased portion alone was removed leaving obviously involved lung behind (Fig 3, 1). These patients have done surprisingly well, having been relieved of fever and other symptoms of toxicity. Almost all have negative sputum by smear and culture.

TABLE II

INDICATIONS	NUMBER OF RESECTIONS*
Tuberculoma	9
Bronchiectasis	8
Bronchostenosis	12
Tuberculous bronchitis refractory to treatment	1
Thoracoplasty failure	28
Lower lobe cavity	6
Destroyed lung	8
Destroyed lung with empyema	4
Thick walled or giant cavity	6
Incapable lung requiring decortication	2
To preserve function	28
Suspected carcinoma	3
To avoid deformity in adolescent	1
Patient refused thoracoplasty	2

*More than one indication may have been present for a single resection

Endotracheal anesthesia supplemented by procaine locally was employed for resection. Intubation was not performed for thoracoplasty. The anesthetic agents most frequently employed were Pentothal Sodium, nitrous oxide, and cyclopropane. Patients were mildly premedicated, and during anesthesia were maintained in as light a plane as possible. Hypoxia and retention of carbon dioxide were prevented by actively assisting respiration. Endotracheal aspiration and inflation of the collapsed lung were frequently carried out. Early in this series, the lateral decubitus position was employed for resections. Three operative spreads of disease occurred in this group of 27 cases. Later, the prone position was employed routinely with no spreads in 80 resections. Postoperatively, the patient was encouraged to cough. Occasionally endotracheal aspiration and, rarely, bronchoscopy were necessary to remove secretions.

In most instances, the amount of lung tissue to be resected was determined at the time of thoracotomy by palpation. Employment of segmental and wedge resection either alone or in combination with other procedures enabled the surgeon to preserve a maximum of functional tissue. When tuberculous bronchostenosis or bronchiectasis was known to be present the involved portions of lung were removed, regardless of their appearance at operation. Routine preoperative bronchography was not performed.

Postresection thoracoplasty was performed after each pneumonectomy, and after subtotal resection, if the remaining lung would not fill the hemithorax when the intrapleural pressures had been reduced to minus 10 to 20 cm. of water. All dead space, especially if bronchopleural fistula were present, was

TABLE III POSTOPERATIVE TUBERCULOUS COMPLICATIONS

	NUMBER OF PATIENTS	PER CENT OF RESECTIONS
Early complications		
Pronopneural fistula	6	3.6
Pronopneural fistula and empyema	2	1.9
Compensatory spread	3	2.8
Late complications		
Infection	3	2.8
Infection of bronchial stump	1	.7

eliminated as soon as possible (usually in three weeks) by thoracoplasty. It is believed that the low incidence of empyema (Table III) in this series is directly attributable to this principle, which was stressed by Graham¹⁸ in 1933.

Every patient in this series received either streptomycin or dihydrostreptomycin for at least a few days prior to operation and a month following the last operative procedure. After the latter portion of 1949, para-aminosalicylic acid was given with streptomycin. Approximately one-fourth received less than ten days of streptomycin therapy prior to resection. An additional one fourth had less than 120 days, and all but 4 patients had received less than 240 days of

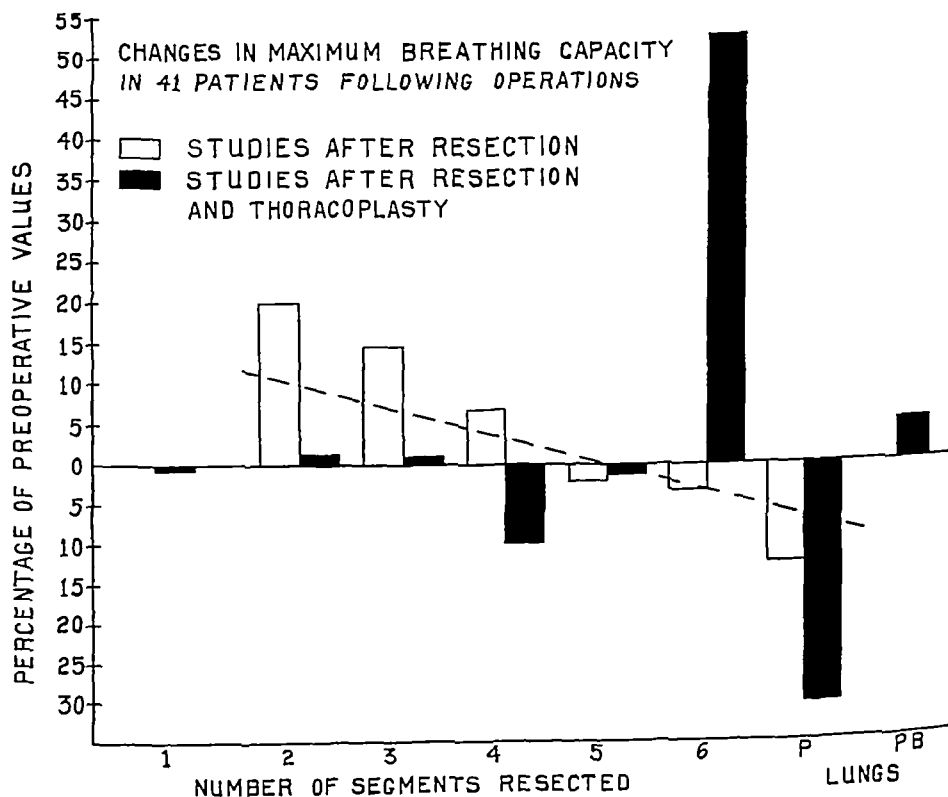


Fig 2

therapy prior to operation. Various schedules and dosages were employed according to contemporary practice. Very few patients had long, uninterrupted courses of streptomycin prior to operation. Streptomycin sensitivity studies were not carried out routinely. However, all patients undergoing operation were given the antibiotic. Our impression that this practice is of value appears to be shared by others.¹⁹

Ventilatory studies were performed preoperatively and postoperatively on 41 patients. The results are summarized in Fig 2. There was a fairly definite correlation between the number of pulmonary segments removed and postoperative change in maximum breathing capacity, as indicated by the slope of

the broken line. When more than 4 segments were resected, the maximum breathing capacity dropped below the preoperative level. When 2, 3 or 4 segments were resected, there was definite improvement in the maximum breathing capacity in comparison with the preoperative determinations. When partial resection and thoracoplasty were performed the final maximum breathing capacity was not markedly changed from the preoperative values. There was one notable exception, as seen in the chart, where 6 segments were resected and subsequent thoracoplasty resulted in marked improvement of respiratory function. The diminution of function was greatest in the pneumonectomy-thoracoplasty group, as seen above *P* in the chart. An exception to this rule was noted in the bronchiectatic cases (*P B* in the chart), where slight but definite improvement in function was found following pneumonectomy and thoracoplasty.

Tuberculous complications are listed in Table III. Bronchopleural fistula occurred in 8 instances, 1 following pneumonectomy, 2 following lobectomy and 5 following segmental or wedge resection. Thoracoplasty was performed in 6 instances with resultant cure of fistulas. The remaining 2 patients developed empyema and 1 of these died. Spread of disease occurred in 3 instances. These, together with 3 late reactivations, responded favorably to streptomycin therapy. Ulceration of the bronchial stump occurred in 4 instances, all of which had preoperative bronchostenosis. This incidence of 33.3 per cent ulceration in the bronchostenotic group was not serious, however, as the lesions responded readily to streptomycin therapy.

The effect of prolonged (and usually intermittent) preoperative streptomycin therapy on complications is analyzed in Table IV. Although the results of such a small series cannot be taken as statistically significant, there appears to be some evidence that prolonged intermittent preoperative antibiotic therapy had a deleterious effect in every category. This may be an unfair conclusion in the light of the fact that those patients receiving long courses of streptomycin were inclined to have more extensive and more serious disease than those receiving short courses of streptomycin.

The effect of postresection thoracoplasty is similarly analyzed in Table V. Thoracoplasty did not improve the results in any category except bronchopleural fistula, when it was curative in every instance in which it was employed.

TABLE IV. EFFECT OF PREOPERATIVE STREPTOMYCIN ON COMPLICATIONS

	PER CENT OF RESECTIONS RECEIVING MORE THAN 90 DAYS OF STREPTOMYCIN PREOPERATIVELY	PER CENT OF RESECTIONS RECEIVING LESS THAN 90 DAYS OF STREPTOMYCIN PREOPERATIVELY
Early complications—		
Bronchopleural fistula	6.1	5.4
Bronchopleural fistula and empyema	2.0	1.8
Empyema spread	4.1	1.8
Late complications—		
Ulceration	11	1.8
Ulceration of bronchial stump	11	2.0

TABLE V RELATIONSHIP OF COMPLICATIONS TO RESECTION WITH AND WITHOUT THORACOPLASTY

	PER CENT OF RESECTIONS WITHOUT THORACOPLASTY	PER CENT OF RESECTIONS WITH THORACOPLASTY
Early complications		
Bronchopleural fistula	7.6	0.0
Bronchopleural fistula and empyema	1.3	3.8
Postoperative spread	0.0	11.5
Late complications		
Reactivation	0.0	3.9
Ulceration of bronchial stump	0.0	5.2

Nontuberculous complications are listed in Table VI. There were 13 patients in this group. Although some of the complications were serious, the responses were favorable with appropriate treatment.

TABLE VI NONTUBERCULOUS COMPLICATIONS

	NUMBER OF CASES	REMARKS
Wound infection	4	This does not include empyema
Atelectasis	3	All responded to aspiration or bronchoscopy
Bronchopneumonia	1	Responded to treatment with antibiotics, apparently not tuberculous
Hemolytic icterus	1	This occurred twice in the same patient, extensive studies failed to reveal the etiology
Postoperative hemorrhage	1	Chest was reopened, severe bleeding apparently emanated from a bronchial artery
Clotted hemothorax	1	Responded to treatment with streptokinase and streptodornase
Toxic psychosis	1	This occurred immediately postoperatively, patient completely recovered
Accidental phrenicotomy	1	

There were 3 deaths in the series, which placed the operative mortality at 2.8 per cent and the case mortality at 3 per cent. The causes of death are listed in Table VII. In the case listed as cardiac arrest, there were some significant observations which deserve comment. The anesthesiologist noted that respirations ceased before the heart stopped beating. This patient was known to have a very flexible chest wall and compressible sternum, apparently produced by a previous thoracoplasty. When she was placed in the prone position and the chest opened, there was a close approximation of the sternum to the vertebral column. Because of the development of respiratory and cardiac difficulties, the patient was turned from the prone to the lateral position. In spite of all the usual and accepted measures directed at resuscitation, the patient died. A

TABLE VII CAUSES OF DEATH

CAUSE	NUMBER
Cardiac arrest	1
Thrombosis of pulmonary artery	1
Empyema and bronchopleural fistula	1
Total	3

pneumonectomy had been planned, and therefore, for statistical purposes the case is listed as a pneumonectomy. However, the hilar dissection was scarcely started when it became necessary to abandon the attempt at lung removal. In the next case, the thrombosis of the pulmonary artery was noted at operation. The patient died twenty-four days later of progressive respiratory insufficiency, presumably due to extension of the thrombus into the contralateral lung. The death from emphysema and bronchopleural fistula occurred in a patient three and one half months postoperatively. Even though no attempt has been made to classify the patients relative to their expected ability to withstand operative procedures, it must be admitted that the last 2 patients were desperate risks. Both had long-standing and extensive disease with large cavitations and had received long courses of streptomycin, which probably had produced resistant organisms.

The present status of the 101 patients is summarized in Table VIII. Fifty-two patients now have stable roentgenograms and negative sputum (by concentrated smear and culture). There have been no late deaths. Information on 6 patients is not sufficiently recent to afford accurate classification although, when last heard from, all were well and had stationary roentgenograms and negative sputum.

TABLE VIII PRESENT STATUS OF 95 SURVIVING PATIENTS

TIME SINCE OPERATION	TOTAL			
	1 TO 4 YEARS	5 TO 9 YEARS	10 TO 19 YEARS	20 YEARS OR MORE
Negative sputum and roentgenograms	—	8	2	82
Roentgenograms and/or negative sputum and/or roentgenograms	—	1	1	10
Re-evaluate follow up	1	2	1	0
Alive	—	—	—	79
Deceased	—	7	2	7
Activity undetermined	1	2	1	0

CASE REPORTS

Case 1—R. B., a 44-year-old Negro woman was known to have a lesion in her left lung. She refused hospital treatment at first but later was admitted to a state sanatorium. She was treated for about five months. Sputum was negative. She left the sanatorium again at which time she was readmitted in April, 1949, because of pleuritic pain on the left. For two days she had signs of bronchial involvement on the left, and streptomycin (2 gm. daily) was given for 2 months and then 1 gm. twice weekly for 3 months. Pulmonary function returned in November, 1950, for the purpose of having a resection. Her left lung was found to be normal on culture from December, 1950, to March, 1951. Roentgenograms on re-examination showed only slight nodules in the upper portion of the left lung, with a cavity in diameter. Respiratory function in April, 1951, was still normal. The patient was discharged in April, 1951, and maximum freedom of activity was maintained. A resection was performed on April 10, 1951. This was followed by a period of 14 months and postoperative course of the patient was excellent.

large cavity, a wedge resection of a 3 cm tuberculous abscess of the lower lobe and excision of 2 small pleural abscesses. Postoperatively, the patient had an uneventful course. X-ray pictures were satisfactory, and sputum remained negative. On Sept 13, 1951, vital capacity was 2.6 L and maximum breathing capacity was 92.2 L per minute. She left the hospital against advice on Nov 16, 1951. Examinations since that time have revealed no evidence of exacerbations of disease.

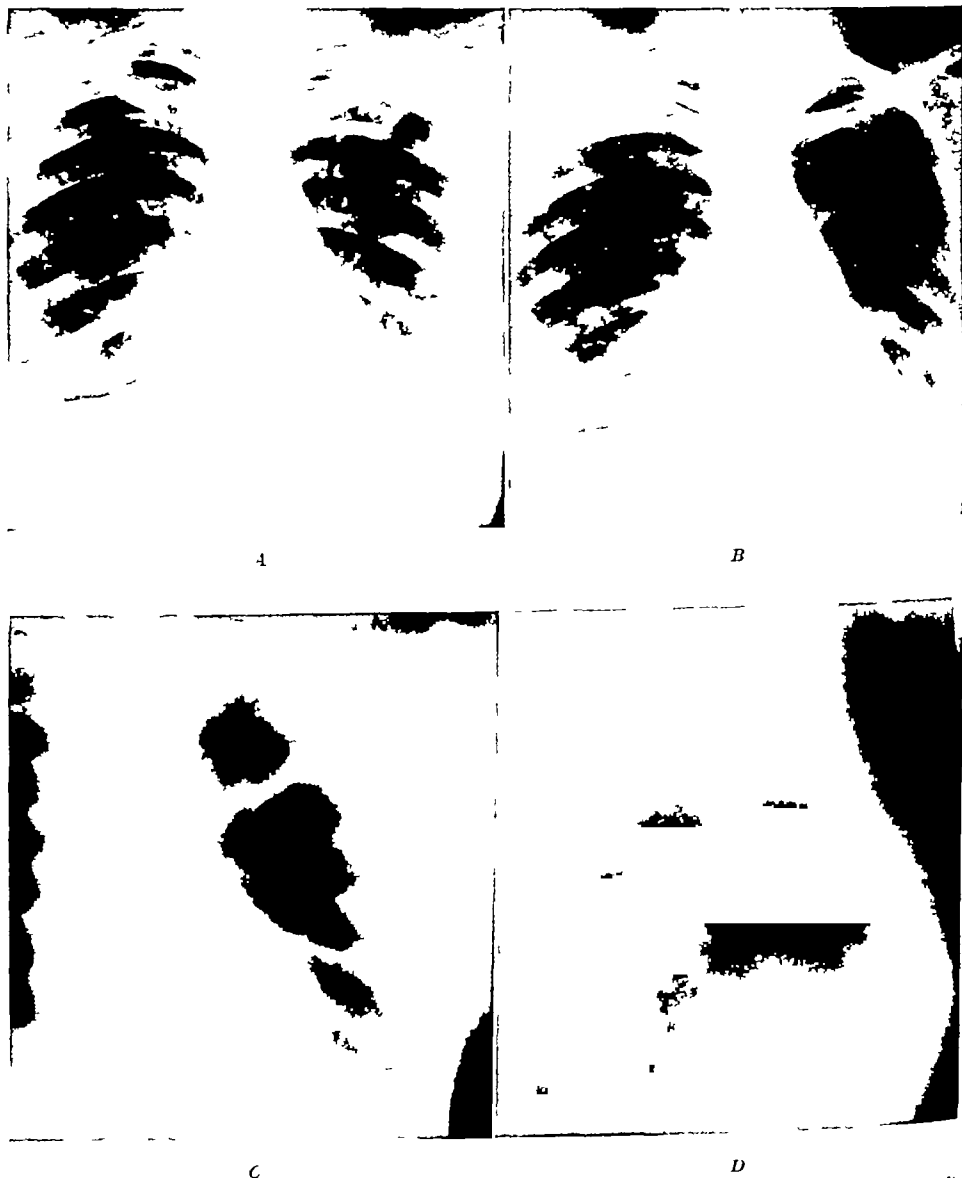


Fig 3 (Case 1) — *A* Preoperative roentgenogram of chest showing mottling chiefly on left and large cavity at left apex.

B Postoperative roentgenogram showing good expansion of remaining portions of left lung and no definite signs of active intrapulmonary disease.

C Anteroposterior laminagram (preoperative) outlining clearly the large cavity in the upper portion of left lung.

D Left lateral laminagram showing the apicoposterior location of the intrapulmonary cavity.

CASE 2—A P, a 27 year old white woman had a diagnosis of pulmonary tuberculosis made in 1944. Sputum was positive for acid fast bacilli, and x ray examination showed evidence of bilateral pulmonary involvement with cavitation on the right. Right pneumothorax was tried but was not successful. Two temporary phrenic operations on the right were performed on Aug 31, 1944, and March 1, 1945. On March 5, 1945, pneumoperitoneum was started but immediately discontinued because of unsatisfactory clinical response. Following long continued bed rest, the patient improved and was discharged in May, 1946. While at home on a modified rest regime, she had a relapse of her disease with positive sputum. She



C

1. Preoperative roentgenogram of chest showing bilateral multiple lesions in both apical regions.
 2. Preoperative roentgenogram showing relatively clear lung fields bilaterally and good expansion of lungs.
 3. Preoperative fluorogram showing multiple lesions in both apices and a suggestive rarefaction within the left lung focus on the left (first anterior intercostal space).

was readmitted to the sanatorium on May 12, 1948. X-ray examination showed increase of disease in the left lung. Streptomycin, 1 Gm daily, was started on July 27, 1948, and continued for 45 days. Roentgenograms showed improvement, and she was started on graduated exercise. In July, 1949, another relapse occurred with cavity appearing in the right lung and sputum again became positive. After further rest, the cavity closed, and sputum converted to negative. In April, 1950, she again started exercise, which was followed by another relapse in August, 1950. On Aug 29, 1950, streptomycin, 1.25 Gm. daily and para-aminosalicylic acid, 10 Gm daily were started.

Because of repeated relapses of bilateral pulmonary disease, the patient was transferred to surgery. Preoperative roentgenograms (see Fig 4, A and B) showed infiltration in both apical regions of the lungs, and the laminagram showed suggestive evidence of a small rarefaction in one of the densities on the left. The first segmental resection was carried out on the left lung on Nov 10, 1950. This consisted of excision of the apical and posterior segments. Examination of the specimen showed several conglomerate tubercle formations and some liquefaction in the center of one of the larger masses.

The patient's last positive sputum (on culture) occurred on Nov 6, 1950. Streptomycin and para-aminosalicylic acid, which had been started in August, 1950, were discontinued on Dec 12, 1950. Because of the presence of lesions in the right lung, the patient was allowed a period of rest and then started on a final course of streptomycin and para-aminosalicylic acid on Feb 9, 1951. This was continued until March 13, 1951. On Feb 13, 1951, an operation consisting of excision of the apical and posterior segments of the right upper lobe was performed. Upon examination, several small caseous foci were found, but no cavity was present. Postoperatively, the patient's convalescence was favorable, and she has remained well.

CONCLUSIONS

A review of the recent literature, as well as our own experiences, reveals that treatment of tuberculosis by resection is not only a safe, but a highly effective, procedure. The early results compare favorably with those of thoracoplasty. Postoperative decrease in ventilatory function is less than that following thoracoplasty since collapse is necessarily greater when the latter procedure is employed as a therapeutic measure. Although a number of years must elapse before this series can be completely evaluated, we have not noted a high incidence of late complications in the earlier cases.

SUMMARY

1. Experiences with 107 resections for tuberculosis upon 101 patients are reviewed.

2. Eighty-two patients (83.7 per cent) have negative sputum and roentgenograms.

3. Morbidity, both early and late, has been relatively slight.

4. There was an operative mortality of 2.7 per cent and a case mortality of 3 per cent.

The authors wish to express their appreciation to the Medical Staffs of Cedarcrest, Laurel Heights, Uncas on Thames and Undercliff Sanatoria for the part they have played in the treatment of these patients, and to Dr Phyllis C. Frost, who skillfully administered anesthesia in almost all of these operations.

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LOCALIZED PULMONARY HYPERTROPHIC EMPHYSEMA

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LOCALIZED pulmonary hypertrophic emphysema is an extraordinarily rare type of cystic disease of the lung. In several recent articles, clinical, radiologic, and pathologic data have been published which indicate that the disease is a definite entity and that it is congenital in origin. The radiologic features follow a rather easily recognized pattern, and permit the diagnosis to be made with considerable accuracy. In the five articles on this subject previously published, ten examples of the disease have been described. The two adult patients in this group are not entirely acceptable for consideration, in one of these¹ the disease was an incidental autopsy finding, and in the second patient² there was associated sarcoidosis of the lung and hilar glands. All of the eight remaining cases³⁻⁵ occurred in infancy or early childhood. These data indicate that the disease is not compatible with long survival, and that operative treatment should be instituted promptly when the diagnosis is made. In the following report another infant having this disease will be described. The patient was subjected to lobectomy, two episodes of cardiac arrest occurred during the procedure, but the patient made a good recovery.

REPORT OF CASE

This infant presented symptoms of asthma, and this disease was present in other members of his family. When first seen the patient had a superimposed acute laryngotracheobronchitis, and this served further to confuse the diagnosis. The chest roentgenogram suggested obstructive emphysema, and a bronchoscopic examination was made in search of a foreign body. Marked emphysema limited to the middle lobe of the right lung was found at operation.

A poorly developed, malnourished, 7 month old male infant, who was dyspneic from birth, was admitted to the hospital* on Dec. 26, 1948, because of cough, dyspnea, and cyanosis. The history recorded that wheezing and dyspnea had been present since birth, and that a diagnosis of bronchial asthma had been made. Symptomatic relief had been obtained with Adrenalin on several occasions. The infant's temperature on admission was 102° F., the chest was barrel shaped, respiration was rapid, with audible inspiratory and expiratory wheezing. There was moderate inspiratory retraction at the costal arch and jugular notch. The admission diagnosis was acute tracheobronchitis and bronchial asthma. A chest roentgenogram demonstrated hyperaeration on the right with partial atelectasis of the lower lobe. The heart and mediastinum were displaced to the left (Fig. 1). The lateral view demonstrated increased anteroposterior diameter of the chest (Fig. 2).

The signs of tracheobronchitis subsided promptly, but the wheezing, dyspnea, and roentgen findings remained unchanged. At this time it was believed that we were dealing with some unusual manifestation of tracheobronchitis, and that extrinsic pressure from

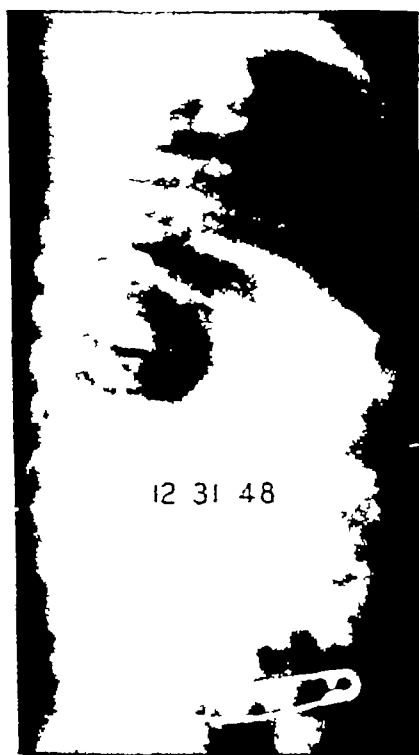
From the Surgical Department, Binghamton City Hospital.
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*Service of Dr. E. R. Dickson.



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Fig. 1—This anteroposterior roentgenogram demonstrates hyperaeration and decreased lung markings on the right with mediastinal displacement to the left. The partially atelectatic right lower lobe is clearly seen. The right hemidiaphragm is depressed and the interspaces are widened.



12 31 48

Fig. 2—Lateral roentgenogram of the chest, anteroposterior view, demonstrating the right lower lobe atelectasis.

enlarged bronchopulmonary glands, or, possibly, a foreign body was producing obstructive emphysema. A bronchoscopic examination was made, diffuse inflammation of the tracheobronchial tree was seen, and no foreign body was found. Following this procedure an acute exacerbation of the patient's symptoms occurred within forty-eight hours. The infant's respiratory distress became extraordinarily severe, his temperature fluctuated between 100° F and 105° F, and for a time it appeared certain that he would not recover. At the end of one week aureomycin was administered, remarkable improvement followed almost immediately, and the infant's temperature was normal within twenty-four hours. Two weeks later the patient was discharged, at this time the roentgen findings were unchanged, and he continued to present symptoms and signs of asthma.

Following his release from the hospital the infant's condition remained stationary. He was readmitted five months later for a period of one week, at which time he had an acute upper respiratory infection with exacerbation of his symptoms. A tuberculin test was negative, and he gave a one plus reaction to several allergens. The chest roentgenogram

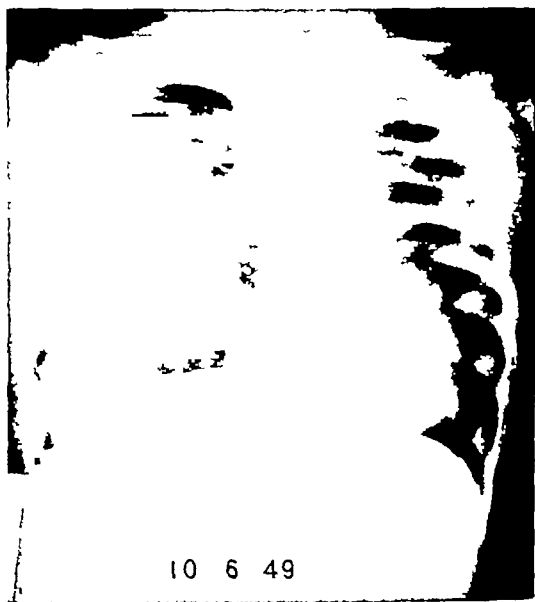


FIG 3.—This preoperative roentgenogram was exposed ten months after the study illustrated in Fig 1. The emphysema is more extensive and increased collapse of the lower lobe of the right lung has occurred. The mediastinum is now widely displaced to the left.

was identical in appearance to that seen during his original admission. At this time a diagnosis of cystic disease of the lung was made. An operation was done on Oct. 11, 1949, under intratracheal ether oxygen anesthesia, the patient was then 17 months old. The preoperative chest roentgenogram is shown in Fig 3.

Operation.—At operation the enormously enlarged emphysematous right middle lobe bulged out of the wound as the pleura was opened. The upper and lower lobes were almost completely atelectatic. The vessels and bronchus of the middle lobe were elongated, and lobectomy was easily done by individual ligation technique. The patient's condition was excellent up to the time that the lobectomy was completed. Cardiac arrest then occurred, ventilation of the lungs and cardiac massage was immediately done, and vigorous action returned after about one minute. Drains were placed and closure was started when cardiac arrest again occurred. Resuscitation for about one minute was again followed by recovery, the operation was then completed without incident.

*Gross Pathology**—The specimen measured 11 by 8 cm. The surface was pale white, and was studded with numerous subpleural blebs which measured 4 to 10 mm. in diameter. It was feathery in consistency, on section it was spongelike, there was no gross abnormality of the larger bronchi (Fig. 4).

Microscopic Findings—The peripheral portion of the lung consisted of large ballooned out spaces produced by intercommunicating alveolar sacs. In places the alveolar ducts up to and including the respiratory bronchioles formed part of the wall of these spaces. These bleblike structures were apparently formed by destruction or absence of interalveolar septa.



Fig. 4.—This roentgenogram of the operative specimen was exposed about one hour after its removal. Very little collapse occurred although the bronchus was not clamped and the specimen was not inflated. The density in the hilar area is a hematoma produced by operative trauma. This study actually demonstrates the pathology better than the dissection of the specimen.

Some of these septa revealed marked bulbous thickenings; in others the interalveolar septa were extremely delicate structures lined with flattened cells. Some of the alveolar spaces were filled with precipitated albuminous edema fluid, a few small foci of low grade inflammatory reaction were present. This picture suggests a type of congenital cystic disease of the lung, in which the terminal and respiratory bronchioles do not constitute the major portion of the picture.

Postoperative Course—The patient reacted promptly following the operation, and convalescence was rapid and without incident. The upper and lower lobes expanded perfectly and healed by primary union, and the patient was discharged from the hospital on the 20th postoperative day.

Presented by Dr. J. S. Crowl, Director, Kilmer Memorial Laboratory.

Subsequent Course—Following his discharge from the hospital the patient's barrel chest deformity was rapidly corrected, and he has remained well. He has been followed for over two years and now appears to be normal in every way. A chest roentgenogram exposed fourteen months after operation is reproduced in Fig. 6.

REVIEW OF LITERATURE

Clinical and Radiologic Data—Data referable to the above patient and the other previously published examples of this disease have been assembled in

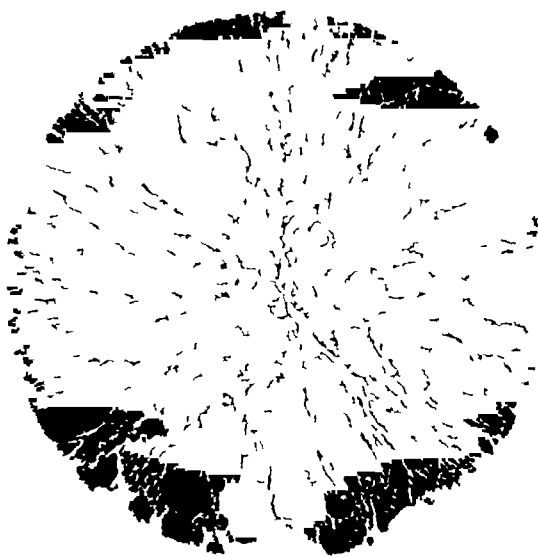


Fig. 5—This photomicrograph demonstrates the extensive pulmonary emphysema.

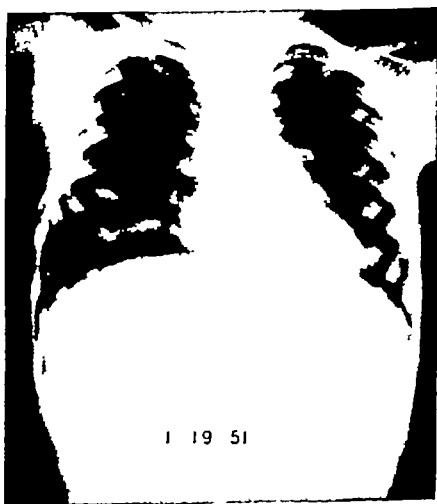


Fig. 6—This negative postoperative chest teleoroentgenogram was exposed ten weeks after operation.

TABLE I

Patient	Date of Birth	Date of Admission	Age at Admission	Sex	Site of Lesion	Pathologic Findings	Treatment	Outcome	Autopsy Findings
Robertson and Jones (5 cases)	1951	Case 1	11 wk	M	Right upper lobe	Dyspnea, cyanosis, external retraction	No examination	Oxygen therapy	Died (autopsy)
			Case 2	M	Right upper lobe	Dyspnea, cyanosis	Typical	1 lobectomy	Recovery
			Case 3	M	Right upper lobe	Whooping, dyspnea, cyanosis	Typical	Needle debridement	Died (autopsy)
			Case 4	M	Left upper lobe	Cyanosis	Typical	1 lobectomy	Recovery
			Case 5	F	Right middle lobe	Cyanosis, whooping	Typical	Aspiration, subcutaneous debridement	Recovery
Shaw (3 cases)	1951	Case 1	1 yr	M	Subapical segment left upper lobe	Whooping	Typical	Segmental resection	Recovery
			Case 2	F	Right middle lobe	Whooping, dyspnea, external retraction	Typical	1 lobectomy	Recovery
Present Case	1952	17 mo	M	Right middle lobe	Whooping, dyspnea, external retraction, attacks of cyanosis	Typical	Associated intral chest diaphragm	1 lobectomy	Recovery

Table I Although they have been included in other reports, neither of the two adult patients appears to be entirely acceptable for study. Excluding these two patients, nine examples, all occurring in infancy or early childhood, remain for analysis. Seven patients were males and two were females, wheezing, dyspnea, cyanosis, and sternal retraction were the outstanding symptoms. The disease was limited to a single pulmonary segment in one instance, in the remaining eight the process was confined to a single lobe. The upper lobes and the right middle lobe were involved with equal frequency, there was no example of the disease occurring in either lower lobe. Chest roentgenograms of eight patients were made. In every instance these studies demonstrated hyperaeration of the affected lung with decreased lung markings, mediastinal displacement toward the contralateral lung, and various degrees of depression of the diaphragm and widening of the interspaces of the involved hemithorax. The roentgenogram of Shaw's Case 2 was identical to that of the patient reported here. In both patients the disease involved the middle lobe of the right lung, and in both the partially atelectatic lower lobe on the same side was clearly demonstrated.

Treatment—Lobectomy or segmental resection without mortality was accomplished in seven patients. One patient, whose chest was deflated with a needle, developed a tension pneumothorax and subcutaneous emphysema followed by a fatality. Thoracentesis was done in another instance with the occurrence of tension pneumothorax. This complication was successfully managed by continuous subaqueous deflation. This patient was subsequently subjected to lobectomy and recovered. This experience should condemn aspiration as an ineffectual and dangerous procedure in this disease.

Pathology—Surgical specimens or autopsy material has been available in all of the patients under consideration. The gross appearance of the involved lung was remarkably uniform. Its surface was white or pale pink, with numerous subpleural emphysematous blebs, on palpation it was feathery, and on cross section spongelike in consistency. At operation the diseased area usually filled the entire hemithorax, and frequently bulged out of the wound when the pleura was opened. Uninvolved lobes on the affected side were partially or completely atelectatic. The vessels of the involved lobe or segment were smaller and longer than average. Following resection excellent expansion of the compressed lobes occurred.

Inflammation was notably absent in the microscopic studies. The alveoli were distended, septa were frequently ruptured, and the alveolar walls were usually thin. Collapsed bronchi with areas of atelectasis alternating with distended air sacs were present in one specimen.³

Etiology—The onset of symptoms at, or shortly after birth, and the roentgenographic evidence of extensive disease in the first weeks of life, together with the absence of inflammation in the surgical and autopsy material, all strongly indicate that this disease is congenital in origin. Anatomic anomalies which might place this disease in the category of an acquired or developmental defect have been proposed. A valve mechanism produced by a fold of mucous membrane in the stem bronchus of the involved lobe was believed by Roves¹ to be the etiological factor in his case. Robertson and James⁴ also found such a

told in one of their patients. In another instance the latter authors believed that a thin walled bronchus might have been compressed by a large adjacent vein. Shaw found unusually flaccid bronchi in his operative specimens, and has suggested that a localized bronchial chondromalacia might be the underlying pathology. An excellent discussion and review of the literature relevant to this aspect of the disease are included in the article of Leahy and Butsch.³

SUMMARY

Lobectomy in an infant having massive hypertrophic emphysema of the middle lobe of the right lung is reported. Two episodes of cardiac arrest occurred during the operation, but the patient made a good recovery. The literature relevant to this disease is reviewed, and the clinical, radiologic, and pathologic data are summarized. All of the nine well-documented examples of the disease thus far published have occurred in infancy or early childhood. Among these, seven were subjected to lobectomy or segmental resection without mortality. The available information indicates that the disease is a clinical entity of congenital origin, and that resection should be done promptly when the diagnosis is made.

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ACUTE TUBERCULOUS FIBRINOUS BRONCHITIS

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FIBRINOUS bronchitis comprises a generic group of bronchial inflammatory diseases which has as its characteristic clinical feature the formation of bronchial casts. The expectoration of casts of the bronchial tree is such a singular clinical phenomenon that the disease has been recognized since the time of Galen. In spite of its long history, only about 200 cases have thus far been reported.¹ In the majority of these patients the bronchial inflammation has been produced by one of the common pyogenic bacteria, examples of the disease due to *Mycobacterium tuberculosis* are exceptional. In the following report a patient having fibrinous bronchitis of tuberculous etiology will be described. This patient presented the clinical picture of severe anoxia due to bronchial obstruction. At bronchoscopy inspissated tenacious bronchial casts were found, the removal of which required a foreign body forceps. Tracheotomy was necessary and the patient subsequently developed a bronchial stricture and bronchiectasis which required pneumonectomy.

REPORT OF CASE

A 38 year old woman, who had had tuberculous cervical adenitis twelve years previously, developed wheezing and a dry cough following an upper respiratory infection. A diagnosis of bronchial asthma was made, chloramphenicol and antihistamine therapy were administered without benefit. Paroxysms of coughing with dyspnea and cyanosis developed within a few weeks, with progression in their frequency and severity. Ten weeks later the patient was hospitalized after a particularly severe exacerbation of these symptoms.

On admission to the hospital on March 11, 1951, the patient's temperature was 101° F, the pulse rate was 120, the respirations were 40, she was pale, and her fingernails were definitely cyanotic. In addition, she was weak, hoarse, and had a dry, brassy nonproductive cough. Physical examination demonstrated no significant findings other than distant breath sounds over the entire left lung. The white blood cell count was 21,000, with 78 per cent neutrophils, the red blood cell count was 4,330,000, the hemoglobin was 78 per cent, the urine contained a trace of albumin. A chest roentgenogram was negative (Fig 1). The admission diagnosis was bronchial adenoma.

Two days following her admission, the patient had a severe coughing paroxysm followed by alarming dyspnea and cyanosis which was not relieved by oxygen. Bronchoscopy was done as an emergency measure. Catarrhal inflammation of the larynx and trachea was present, and the left main bronchus was found to be almost occluded by tenacious, firm, grayish white, inspissated material. This could not be aspirated, and it was laboriously removed in small fragments using a foreign body forceps. After the removal of the cast, the bronchial mucous membrane was seen to be intensely inflamed, it bled on contact with the aspirator. At the end of the procedure the patient's anoxia was relieved, she then sat up on the operating table, coughed effectually, and raised several additional pea sized frag-

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From the Surgical Departments of Our Lady of Lourdes Hospital Binghamton N. Y. and Charles S. Wilson Memorial Hospital Johnson City N. Y.

status of identical material. During the next twenty four hours a recurrence of the alarming respiratory distress required a second bronchoscopy with removal of inspissated secretions, this procedure was followed immediately by a tracheotomy.*

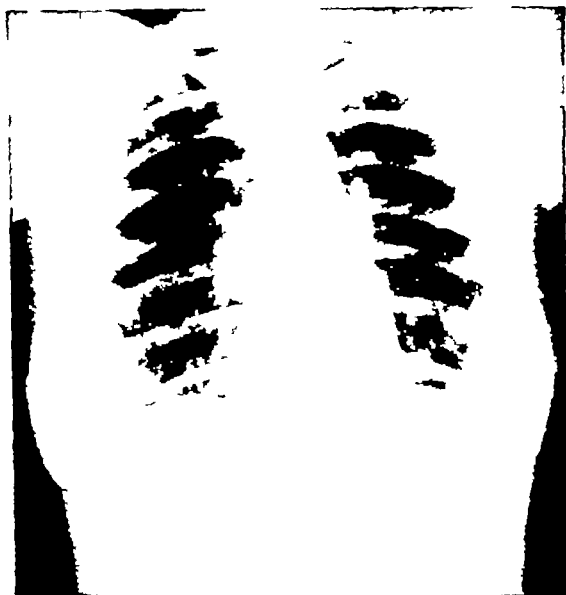


Fig. 1—This chest roentgenogram was exposed a few hours before the first bronchoscopic procedure. Both lungs are well aerated although signs of obstruction of the left main bronchus were present. This study was believed to be negative but in retrospect the prominent left hilum shadow suggests the presence of disease in the superior segment of the lower lobe.

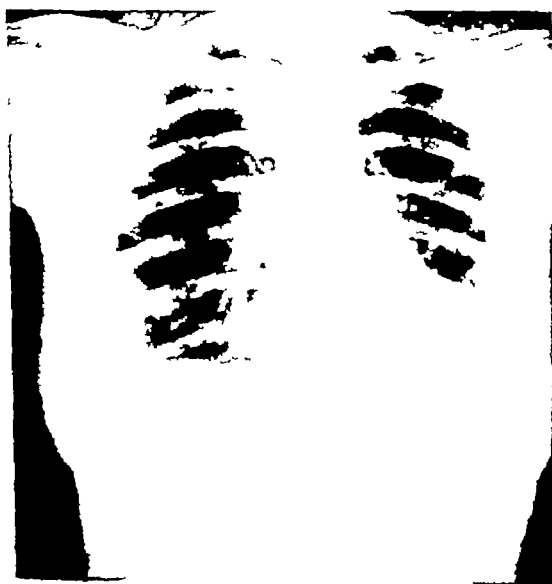


Fig. 2—This roentgenogram was exposed ten days following the first bronchoscopic procedure and tracheotomy. A bronchopneumonic infiltration is seen on the left lateral studies and a consolidation in the lower lobe.

By Dr. Manuel M. Monasterio

Postoperatively, frequent tracheal aspirations with a prostatic type catheter were effectual in clearing the tracheobronchial tree. The septic fever continued, and the chest roentgenogram demonstrated a scattered infiltration in the lower lobe of the left lung (Fig 2).

A specimen of the material collected at bronchoscopy for culture was erroneously submitted for biopsy. It contained gram positive cocci and gram negative bacilli, no acid fast organisms were found. Secretion aspirated after tracheotomy yielded a growth of several pyogenic organisms. Antibiotic therapy with penicillin was started, no response was obtained, and this agent was followed by terramycin. The patient improved following administration of the latter antibiotic, but she did not become afebrile until three weeks after the operation. At this time the tracheotomy tube was removed and the patient was discharged from the hospital, although the infiltration in the left lung was not entirely resolved.



Fig 3—This photograph of the left lung demonstrates the inflammation of the bronchi and the extensive suppurative and caseous disease in the lower lobe. The bronchial disease has not progressed to the stage of stenosis and the narrowing of the main bronchus is much less prominent than would be expected from its appearance at bronchoscopy.

Following release from the hospital the patient failed to make satisfactory progress. The tracheotomy sinus did not heal, the septic fever recurred, she again developed a severe brassy cough and raised a large amount of purulent sputum. The inflammatory process in the lower lobe of the left lung did not resolve, and a new area of infiltration appeared in the upper lobe. Sputum cultures were made, and eight weeks later one of these was reported positive for tuberculosis. Numerous additional cultures were negative before this finding was confirmed. A bronchoscopic examination at this time demonstrated

acute inflammation and marked narrowing of the left bronchus. It was then obvious that severe bronchiectasis was also present, and the patient was accordingly prepared for pneumonectomy. A good remission of symptoms followed six weeks' therapy with PAS and streptomycin. Pneumonectomy was done on Oct. 30, 1951, under intratracheal ether oxygen anesthesia using the prone position. A bronchoscopic aspiration was done at the end of the procedure.

Forty-eight hours after operation the patient began to develop slight hoarseness and difficulty in raising bronchial secretions, which were unusually viscid. Progression of these symptoms occurred during the next twenty-four hours, and the patient required oxygen. A tracheotomy was then done, a large amount of ropy, viscid, purulent secretion was evacuated, with striking relief. Convalescence thereafter was uninterrupted. The tracheotomy tube was removed after one week, and the patient was discharged ten days later.



Fig. 1.—This roentgenogram was exposed seven weeks after pneumonectomy. A modified thorico-plasty has been done; the right lung is clear.

Gross Pathology.—The main bronchus, its major branches, and even the smaller ramifications were filled with gray, tenacious, purulent material. Both lobes were only partly inflated. The upper lobe in its lower four-fifths was airless and rubbery. The medial portion of the lower lobe was atelectatic and nodular. Multiple discrete grayish, small, whitish nodules, which measured up to 6 mm. were present in both lobes, the largest were located in the upper lobe (Fig. 2).

Endoscopic Findings.—The main bronchus showed absence of epithelium with formation of a thick, inflamed granulation tissue. Bronchi of all sizes were characterized by a hyperplastic infiltration with partial or complete destruction of muscularis with resultant stenosis. In a few of the larger bronchi, rare Langhans giant cells were present in the lumen, but with no granuloma formation.

All sections of the pulmonary parenchyma displayed numerous large and small, hemorrhagic nodules of infiltration, which were composed of infiltrating epithelial cells with

hyperplasia and formation of a thick, inflamed granulation tissue. The tracheotomy tube was used for ventilation with minimal trauma. The patient died of a massive pulmonary embolism. The patient was treated by Dr. Alexander A. K. Smith, Charles S. Wilson Memorial Hospital.

numerous scattered multinucleate giant cells of the Langhans type. In many instances, especially in the larger granulomas, centers of caseous necrosis were prominent. Lymph nodes from the hilar area were likewise involved by an identical process.

Subsequent Course—A two stage thoracoplasty was completed a few weeks later following which the patient has continued to make excellent progress. All postoperative examinations of the sputum have been negative, and the patient is now doing her own housework. A recent chest roentgenogram is reproduced in Fig. 4.

ETIOLOGY

Fibrinous bronchitis has received comparatively little attention in the recent literature, and a review of the subject has not been published since 1920.²⁸ Most of the communications have been isolated case reports, many of which are incompletely documented, a large percentage of these publications has appeared in the foreign literature. Both acute and chronic types of the disease have been described, the acute form is comparatively uncommon. The bacteria which have been cultured from the casts include streptococci, staphylococci, corynebacterium, diphtheria, *Klebsiella pneumoniae*, *Mycobacterium tuberculosis*, and *Hemophilus influenzae*.

A bewildering variety of both thoracic and constitutional diseases has been associated with the formation of bronchial casts. In the pulmonary group are included asthma, influenza, pneumonia, tuberculosis, congestive heart disease, bronchial compression due to any cause, and the inhalation of various noxious agents. The constitutional group includes measles, typhoid fever, scarlet fever, erysipelas, smallpox, pemphigus, and impetigo.

Walker²⁸ expressed the view that the expectoration of casts may be either symptomatic or idiopathic. It was his opinion that fibrinous bronchitis actually is a disease entity, but that this diagnosis should not be made if the formation of the casts were related or secondary to another disease. The association of bronchial casts with various bacteria and many unrelated diseases makes it difficult to accept the concept that fibrinous bronchitis actually is a disease entity.

An allergic basis for the disease has been proposed, and this thesis is supported by its association with asthma and increase in eosinophils.¹⁸ Comparison of the disease with mucous colitis has been made.^{8, 28} A metabolic disturbance of the bronchi¹¹ has been proposed. In addition, it has been suggested that the disease is a type of neurosis² or that some infectious, chemical, or physiochemical agent is the responsible etiologic factor.²³

From the above it is seen that a satisfactory explanation for the occurrence of these casts has not yet been advanced. It does seem clear, however, that the acceptance of fibrinous bronchitis as a disease entity is at best a tenuous concept. At this time it appears acceptable to consider these casts as a clinical phenomenon due to an abnormal response of the bronchial glands, and epithelium to a bacterial, thermal, or chemical irritant. Clinically, the formation of bronchial casts appears to occupy a position somewhere between diphtheritic membranes and the bronchial crusts which may form after tracheotomy in

patients having acute tracheobronchitis. In these diseases extensive destruction of epithelium in the former and the drying effect of tracheotomy in the latter appear to be the important etiologic factors.

THE BRONCHIAL CASTS AND ASSOCIATED BRONCHITIS

The physical properties of bronchial casts are fairly uniform. They are white or grayish-white in color, rather firm and rubbery in consistency, they are occasionally blood stained.¹² In typical examples the larger branches are tubular, the smaller branches are usually solid.^{6, 10, 23} Microscopic studies demonstrate a mucous or gelatinous matrix with interlacing fibrils of fibrin. The casts commonly contain epithelial cells, red blood cells, leukocytes, and various bacteria. Descriptions of the associated bronchial pathology vary considerably, reflecting the infrequent autopsy and bronchoscopy studies that have been made. Loss of epithelium,²³ breaks in epithelium, and excessive numbers of mucus-secreting cells of the bronchial glands⁶ have been described. Callahan⁶ reported that bronchoscopy showed his patient to have normal bronchi ten days after the expectoration of casts.

TUBERCULOUS BRONCHITIS

Although this article is concerned with a tuberculous variant of fibrinous bronchitis, it seems appropriate to summarize those features of tuberculous bronchitis which are germane to this discussion. By contrast, tuberculous bronchitis is a common disease. In a recent article 500 cases were reported by a single observer.¹² A voluminous literature on this subject has accumulated since 1931 when Elloesser¹⁰ called attention to the importance of tuberculous bronchial stenosis. It is now well established that tuberculous bronchitis is a common complication in patients having advanced pulmonary tuberculosis. In exceptional instances bronchial disease may follow the rupture of a caseous lymph node into the tracheobronchial tree.^{3, 4} Examples of tuberculous bronchitis in patients having little or no pulmonary tuberculosis are comparatively rare.^{1, 2} Bronchial infection has been found to be an important unfavorable factor in the prognosis of pulmonary disease, and in addition is commonly a serious deterrent to the benefits of collapse therapy.^{1, 2} It has been established that a particularly grave prognosis is occasioned by bronchial structure associated with post-stenotic pulmonary suppuration, and that the most satisfactory treatment for patients in this category is resection.¹ Contrary to what might be expected, specific reference to the association of fibrinous bronchial casts with tuberculous bronchitis has not been found.

NONTUBERCULOUS FIBRINOUS BRONCHITIS

Fibrinous bronchitis may be acute or chronic. In what appears to be an unusual subdivision, a third invariably fatal type has been mentioned in which casts are not expectorated but are discovered at autopsy.² In general, the fatal type is one in which the constitutional symptoms are severe, the course of the disease is brief, the illness terminating either in death from asphyxia or sepsis without recurrence.

Two recently reported examples of the acute form of the disease are of interest. Johnstone's¹⁴ patient was a 21-year old student nurse who presented signs of an acute respiratory infection with fever, tachycardia, and increased respiratory rate. Within twenty-four hours she developed extreme expiratory dyspnea, harassing cough, and cyanosis. This episode was terminated by the expectoration of a large bronchial cast. Signs of anoxia recurred and were progressive, eighteen hours later an emergency tracheotomy was done, and additional casts were removed with forceps. The patient died a few hours later, the duration of her illness was less than two days. The second patient¹⁵ was an infant, 20 months old, whose illness also began with signs of acute bronchitis. Dyspnea, cyanosis, and radiological evidence of atelectasis of the right lung developed within five days. At bronchoscopy fibrinous material occluding the right main bronchus was removed with the aspirator and forceps. The patient recovered rapidly and was discharged nine days later. Comparatively few other publications record the occurrence of atelectasis^{13, 16, 17, 22, 23} or the bronchoscopic removal of casts.^{12, 13}

The chronic form of fibrinous bronchitis is the one most commonly encountered. Examples of this type usually occur in middle life, and the patient generally gives a history of previous attacks of acute bronchitis with chills and fever. Characteristically, the patient has repeated bouts of severe paroxysmal dyspnea and cough which terminate in the expectoration of a bronchial cast. A peculiar inspiratory flapping sound, produced by movement of the partially separated casts, may be heard over the affected lung.²⁰ This physical sign is repeatedly described in the literature, but is rarely heard.

The casts resemble curdled milk in appearance, they are best seen by immersing them in water, in this way branching to bronchial divisions as small as those of the fifth order may be demonstrated. As many as six casts may be expelled in a single day,⁹ and this may continue for weeks or even months. Recovery may be followed by recurrence of the symptoms after an interval of months or years.

TUBERCULOUS FIBRINOUS BRONCHITIS

Information regarding the incidence of fibrinous bronchitis in pulmonary tuberculosis is fragmentary. Norris and Landis²⁰ did not encounter a single case among the patients seen during a thirteen-year period at the Henry Phipps Institute. In addition, they saw no examples at autopsy among 662 individuals having pulmonary tuberculosis. The same authors, however, found twenty examples in the literature, thirteen of which were discovered at post mortem examination. Schroder²⁶ observed five cases among 4,716 tuberculous patients. Auerbach² reported one case observed at autopsy in a patient having extensive chronic pulmonary tuberculosis. The evidence supporting a diagnosis of tuberculosis in the patient reported by Perlstein²² is inconclusive. His patient presented massive atelectasis and immediate improvement followed the expectoration of a bronchial cast. The patient's sputum was negative, the chest

roentgenogram suggested inactive tuberculosis in both upper lobes. One must conclude that fibrinous bronchitis is a rare complication of pulmonary tuberculosis.

DISCUSSION

As previously noted, reference to the association of bronchial casts with tuberculous bronchitis has not been found. It seems likely, nevertheless, that the patient described in this report is an authentic example of tuberculous bronchitis and that this disease initiated the formation of the bronchial casts. The history of tuberculous cervical adenitis and the absence of evidence of parenchymal disease in the admission chest roentgenogram suggests that rupture of a caseous lymph gland into the left bronchus may have initiated the bronchial disease.* The bronchopneumonic lesion in the lower lobe of the left lung appeared immediately following the bronchoscopic procedure and tracheotomy. This sequence suggests that the pulmonary infection was due to partial bronchial obstruction and retained purulent secretions. Similar episodes following diagnostic bronchoscopy occurred in two of the eight patients reported by Ornstein and Epstein.²¹ If a diagnosis of tuberculous bronchitis had been established when the tracheotomy was done streptomycin therapy might have effectually prevented the progress of the disease which eventuated in pneumonectomy.

Considering the length and size of bronchial casts, it is rather surprising that asphyxia or atelectasis has not been more commonly reported. This may be explained in part by the fact previously noted that the larger segments of the casts are hollow, and thus usually fail to cause complete bronchial obstruction. As a consequence, some air can be inspired in the affected lung, the 'behic blast' of the cough mechanism is preserved, and the patient is ordinarily able to expel the cast, although this may be extremely difficult. An explanation is required for the unusual difficulty in expelling casts experienced by patients having the acute form of the disease. This is probably due to an associated severe bronchitis with destruction of bronchial epithelium. Under such circumstances adherent casts and ineffectual cough would be anticipated. Severe inflammation of the bronchial mucous membrane was observed in the patient reported here. The radiological findings are variable²² and the chest roentgenogram is frequently negative, the occurrence of atelectasis has already been mentioned.

A survey of the literature shows that bronchoscopy has been infrequently utilized in either the diagnosis or management of fibrinous bronchitis. The few reports thus far published and the case described here, show the advantages of a bronchoscopic approach to this problem. In any acute respiratory infection which is complicated by the development of signs of bronchial obstruction and which has the possibility of fibrinous bronchitis should be entertained and bronchoscopy performed. The development of massive atelectasis under these circumstances also points to this diagnosis if the patient's history does not suggest one

* A similar case was not observed at operation but microscopical study demonstrated fibrinous bronchitis.

of the conventional causes for this complication. The physical properties of the casts may require their removal under direct vision with forceps. Under such circumstances a tracheotomy might be entirely ineffectual.¹⁴ Recurrence of casts may require tracheotomy, but this procedure should be preceded by their thorough removal by bronchoscopy.

SUMMARY

A patient having bronchial obstruction and alarming anoxia due to acute tuberculous fibrinous bronchitis is reported. Bronchoscopic removal of casts and tracheotomy were required, the patient subsequently developed, in rapid sequence, a bronchial stricture and bronchiectasis. Pneumonectomy was required, the patient recovered. It is emphasized that the occurrence of atelectasis or bronchial obstruction in the course of an acute respiratory infection may be due to fibrinous bronchitis. It is recommended that bronchoscopy should occupy an important place in the diagnosis and treatment of this rare disease which may be rapidly fatal.

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BENIGN THYMOMA IN THE HILUS OF THE LUNG

CASE REPORT

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RECENT reports in the literature^{1, 2} have emphasized the importance of early exploratory thoracotomy for intrathoracic tumors which defy clinical identification. With more widespread acceptance of this surgical philosophy, tumors formerly thought to be very uncommon will undoubtedly be seen more frequently. The case history to be presented is at the present time a rarity, although with the increasing number of diagnostic thoracotomies being performed the true incidence of this type of tumor may be established.

CASE REPORT

E P, a 37 year old white housewife, was referred on Oct 13, 1951, for diagnosis and treatment of a mass in the hilus of the left lung. The lesion was discovered on a routine roentgenogram of the chest. There were no complaints referable to the mass, and no history of cough, hemoptysis, or dyspnea could be elicited. No symptoms were present to suggest myasthenia gravis. The patient recalled having had pneumonia in 1946, and roentgen films taken at that time, which were subsequently obtained (Fig 1, A), showed the presence of the hilar mass. It was suggested that additional chest films be taken one month after recovery but, because of the absence of symptoms, the patient did not return.

Physical examination revealed normal vital signs, and no abnormalities were noted on examination of the lungs and heart. Roentgen films taken at the time of hospitalization (Fig 1, B) revealed a homogeneous, clearly defined, smooth walled oval density in the left lung adjacent to the hilar region. The lateral x ray projection showed that the mass was superimposed on the other structures of the hilus. When viewed fluoroscopically the mass did not pulsate, and no connection to the esophagus could be demonstrated. It moved only slightly with diaphragmatic excursion.

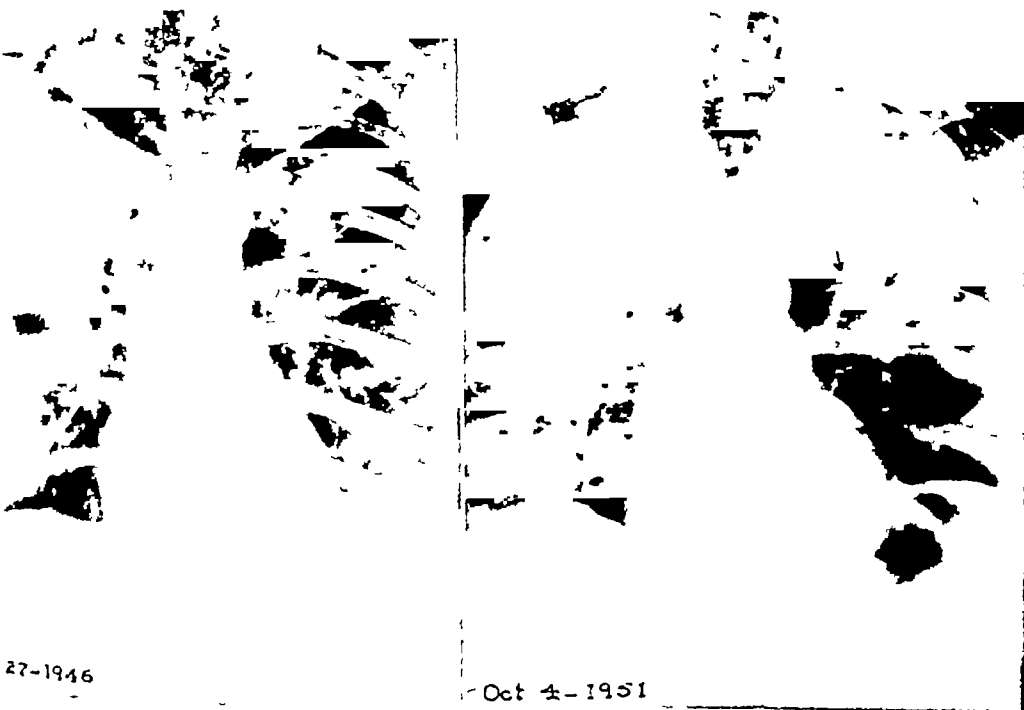
Bronchoscopic examination revealed no bronchial compression nor other abnormalities. Routine laboratory work included urinalysis, complete blood count, sedimentation rate, and serological studies, all of which were normal.

On the presumptive diagnosis of a bronchiogenic cyst, an exploratory thoracotomy was performed on Nov 13, 1951. The left pleural cavity was entered through the bed of the fifth rib which was excised subperiosteally. The tumor, measuring 2 by 2.5 by 4.5 cm., was located in the left hilar region at the base of the interlobar septum. It was covered laterally by visceral pleura, and medially was in close proximity to the pulmonary artery. The parenchyma of the upper and lower lobes covered its superior and inferior surfaces. It was salmon pink in color and was easily shelled out by blunt dissection, being attached only by a small hilar blood vessel which was divided and ligated. Exploration of the left thoracic cavity revealed no further abnormality, and no thymic remnant was noted. After an uneventful convalescence the patient was discharged on the tenth postoperative day.

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PATHOLOGY

The oval mass, which was well encapsulated, measured 4.5 by 2.5 by 2 cm, and on section revealed a homogeneous fleshy, grayish pink surface. Microscopically the mass was quite cellular, and showed a well defined fibrocollagenous capsule. Small lymphocytes (thymocytes) were the predominant cells, and scattered throughout the sections were many Hassel's corpuscles composed of epithelial cells arranged in a concentric fashion. Some of these corpuscles showed central hyalinization. The small lymphocytes were closely aggregated around the corpuscles and formed a follicular arrangement (Fig 2). No evidence of differentiation into cortex and medulla was noted. Arterioles were seen throughout the section, some of which showed hyalinization of the wall, and subcapsular hemorrhage was also noted. No evidence of invasion or malignant degeneration was noted. A diagnosis of thymoma was made pathologically. However, since the tumor probably arose from an aberrant thymic anlage, it could also be called a thymic choristoma.



A

B

Fig 1. A—Note the well circumscribed nature of the left hilar mass.
 Fig 1. B—Roentgen films taken five and one half years later show essentially no change in the left hilar density.

DISCUSSION

To our knowledge, only one other such case has been reported,⁷ the tumor discovered at autopsy. This lesion caused compression of the bronchus, and apparently contributed to the death of the patient during a routine tonsillectomy.

Embryologically the existence of thymic tissue in the hilus of the lung is difficult to explain. A brief review of the development of the thymus in the

human being will readily account for the thymic cell rests that have been reported in the thyroid gland, lateral cervical regions, and in association with the parathyroid glands.^{4, 5} Arey⁶ states that the primordia of the human thymus appear at the end of the sixth week of intrauterine life (12 mm embryo) as ventral pouches of the third pair of pharyngeal pouches. The origin of the parathyroid glands from the dorsal wings of these same pouches explains the occasional existence of thymic cell rests in the cervical region. Other investigators^{7, 8} have reported rudimentary thymic tissue arising from the fourth pharyngeal pouch. These, however, probably do not take part in the formation of the normal thymus gland. The earliest indication of the future respiratory organs is the so called laryngeal-tracheal groove which appears in 3 mm

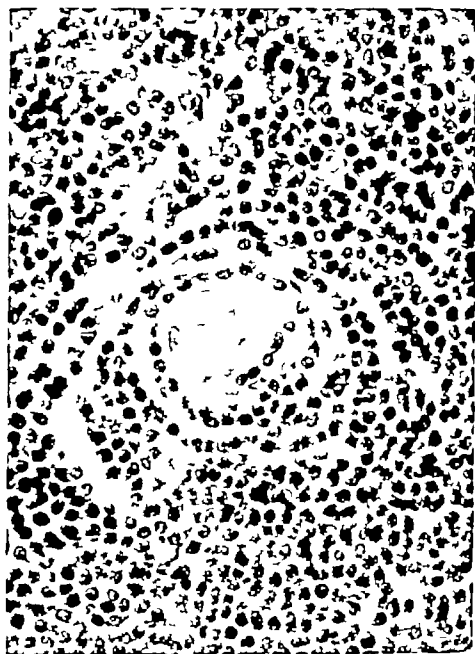


Fig. 2.—This microscopic section shows Hassell's corpuscle with the small thymocytes arranged about it presenting a histologic pattern compatible with the diagnosis of thymoma.

embryos and runs lengthwise in front of the gut just caudad to the fourth pharyngeal pouch. This gives some tenability to Patterson and Heller's³ theory that the presence of ectopic thymic tissue in the lung could be explained by an abnormal intimate relationship between the rudimentary thymic tissue in the fourth pharyngeal pouch and the tracheal anlage, the former descending with the latter.

The existence of thymic tissue in an adult after regression of the normal thymus gland suggests that this should be considered a true tumor arising from an aberrant cell rest. If this were not a neoplasm, one would expect the same factors to act upon it as were in effect on the thymus in its normal location.

The presence of this tumor in a 37-year-old woman without grossly demonstrable persistent thymic tissue and its lack of differentiation into cortex and medulla would favor the diagnosis of thymoma instead of an aberrant cell rest

SUMMARY

An unusual case of a thymic tumor arising in the hilar region of the lung at the base of the left interlobar septum is presented. To our knowledge, this is the second such case to be reported, although it is our feeling that more tumors of this type may be discovered as the number of survey chest films and subsequent thoracotomies increases

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Announcement

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The 33rd annual meeting of the American Association for Thoracic Surgery will be held March 27, 28, and 30, 1953, in San Francisco, Calif. Headquarters will be at the Hotel Fairmont. Suitable accommodations also have been reserved at near by hotels, the Mark Hopkins, the Huntington and the Sir Francis Drake.

Requests for Hotel Reservations

These should be mailed to Dr. H. Brodie Stephens, Chairman of Hotel Committee, American Association for Thoracic Surgery, Room 200, 61 Grove St., San Francisco 2, Calif. The following information must be included: (1) two choices of hotels, (2) type of accommodation desired—single, double, twin, room or suite, (3) name of other room occupants, if any, whether wife or another doctor, (4) date and approximate hour of arrival, (5) date of departure, (6) return address.

Abstracts for Papers

Abstracts for the presentation of papers at the 1953 meeting must be received on or before Dec. 1, 1952, otherwise they will not be considered by the Program Committee. *Five, repeat, five* copies of each abstract should be sent to the Secretary of the Association, Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif. They should not be sent to the Editor.

The abstracts should contain from 200 to 250 words, and should accurately reflect the contents of the completed paper.

Applications for Membership

Applications for Associate Membership in the American Association for Thoracic Surgery must be received by the Membership Committee not later than December 1, 1952, to be considered for action at the meeting in San Francisco in March, 1953. Applications received on a date beyond that deadline must be deferred for consideration until 1954.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the sponsoring statement on the application form, the sponsors must submit a separate letter concerning the applicant direct to the Chairman of the Membership Committee. These regulations are at the direction of the Council and were made effective at the meeting in Dallas in 1952.

Completed application forms should be sent to the Chairman of the Membership Committee, Edward M. Kent, M.D., 1406 Clark Building, Pittsburgh 22, Pa.

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Original Communications

SILICOTIC AND TUBERCULOSILICOTIC LESIONS SIMULATING BRONCHIOGENIC CARCINOMA

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IT HAS long been known that patients suffering from pulmonary silicosis with or without an added tuberculous infection may develop areas of fibrosis which in chest roentgenograms appear as more or less massive areas of density. A number of writers have drawn attention to the fact that these densities may be mistaken for bronchiogenic carcinoma, developing on a background of silicosis. In 1947 Woodruff and Kelley¹ reported a case of tuberculosilicosis simulating bronchiogenic carcinoma and reviewed the literature. They were able to find eight other cases of histologically confirmed silicosis or tuberculosilicosis which had been diagnosed lung tumor erroneously on clinical and radiographic grounds. Their patient showed a unilateral circumscribed density in the right lower lobe with radiographic evidence of a diffuse silicotic reaction; he had a long history of exposure to dust. The case of Bridshaw and Chodoff² had a history of exposure to silica but no evidence of a diffuse silicotic reaction. When their patient was seen first the shadow was unilateral but over a period of fifteen months while he was under observation a density appeared in the opposite apex. In all the other cases reviewed by Woodruff and Kelley the densities were bilateral.

A number of patients have been seen at the Toronto General Hospital with evidence of silicosis who showed massive densities, either unilateral or bilateral, which could be confused with carcinoma, and some of them actually had carcinoma. However, special interest has been aroused by a group of eight patients who, with one exception, have not shown the usual radiographic picture of silicosis, and have presented with a unilateral density of the type usually seen in bronchiogenic carcinoma but due in fact to silicosis or tuberculosilicosis. The eight patients who are the subjects of this report were all men in middle age, seven of the eight complained of symptoms of the type usually associated with bronchiogenic carcinoma. These seven have been

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operated upon, and the diagnosis has been confirmed by histologic study. All have had a history of some exposure to silica dust, but a striking feature is that in some of them the exposure had been relatively short and many years before the onset of symptoms.

The reason for the development of massive pulmonary densities in patients with silicosis has never been elucidated. At the various Saranac Laboratory Symposia on Silicosis there was much discussion of this problem. Gardner² gave it as his opinion that infection, and most often tuberculous infection, is the underlying cause of conglomeration, although associated action of nonsiliceous dusts probably plays an accessory role. He suggested that in simple conglomerate silicosis the infection had healed and played only an etiological role in causing the density, while in tuberculosilicosis the infection is still active and recognizable as a tuberculous process. He gave a very detailed discussion of the various types of reaction which might result depending on whether the infection preceded the dust exposure, was coincident with it, or followed it.

At the Fourth Symposium, Pendergrass⁴ raised the question of whether or not a massive shadow seen in a roentgenogram may be considered due to silicosis in the absence of the classical picture of diffuse nodulation, and stated that in his opinion it could. Gardner⁵ had stated earlier, "The massive type of shadow usually appears upon a background of generalized nodulation, but there are plenty of cases in which this background is lacking or obscured." He pointed out that in such cases examination at autopsy had usually revealed minute nodules in other parts of the lungs. Riddell⁶ described a case in which the exposure to silica had been very heavy, but the radiographs had been diagnosed as indicative of tuberculosis. Autopsy had revealed no evidence of infection. There was a conglomerate focus of silicosis, and a few nodules were discovered in other parts of the lungs. Gardner⁷ ended the discussion by saying, in spite of his statement quoted above, that before a massive shadow, seen in a roentgenogram, could be considered siliceous he must insist on seeing nodular shadows. He admitted that in following this rule mistakes would be made and injustice done to individuals.

This rather brief summary of the various discussions at the Saranac Laboratory Symposia, and in particular the Fourth, is given because, if our interpretation of the pathology is correct, the series of cases to be described below includes examples of massive densities due to both conglomerate silicosis and tuberculosilicosis in the absence of radiographic evidence of nodulation.

CASE REPORTS

CASE 1—Mr. L. D., aged 49 years, was admitted to the Toronto General Hospital on Dec. 18, 1946, with a provisional diagnosis of bronchiogenic carcinoma. He had been well until two years previously when he began to lose energy and tire easily. For eighteen months he had had substernal pain radiating around the right costal margin, aggravated by a deep breath or cough. In June, July, and November, 1946, he had been treated in the hospital for attacks of right-sided pneumonia, and since the first attack in June he had had a cough productive of 15 cc per day of mucopurulent sputum. He had noticed blood in his sputum on two occasions during intervals between his attacks of pneumonia.

The patient weighed 150 pounds in weight felt weak and was short of breath on mild exertion. He was a large man weighing 175 pounds who looked chronically ill. There were no abnormal signs in the chest or elsewhere on physical examination. Roentgenograms indicated partial atelectasis and consolidation of the right middle lobe. There was no evidence of silicosis. A bronchogram showed no filling of the middle lobe bronchus suggesting complete obstruction. Bronchoscopic examination revealed filling of the orifice of middle lobe bronchus with mucoid secretion coming from the middle lobe. Repeated sputum examinations failed to show malignant cells. It was concluded that the patient had obstruction of the middle lobe bronchus probably due to carcinoma, and secondary infection in the lobe.



Fig. 1—A, B. Frontal and lateral views of chest showing middle lobe opacity.

At operation on Jan. 15, 1947, the lung was adherent to the chest wall. When freed, it was a mottled gray-green appearance scattered throughout were firm nodules varying in size from 1 to 3 mm in diameter. The middle lobe was about 50 percent reduced in volume at 4 days gray in color. There was a thin lining of all the hilar structures with lymph nodes adherent to the vessels and bronchi. On chest x-ray, the consolidation was very dense in the middle lobe bronchus and the anterior pulmonary vein came away from the main stem, went in the vein with no filling, severe hemorrhage. A pneumonic

Pathologic Examination—Gross This confirmed the presence of multiple small discrete nodules throughout the lung and partial atelectasis with consolidation of the middle lobe. On section this lobe showed moderate dilatation of the bronchus, and the parenchyma was dark gray with islands of greenish brown tissue. All the structures of the hilus were densely adherent to each other, and the lymph nodes were enlarged, hard, and adherent. The dissection necessary after tearing the pulmonary vein had disturbed the relations about the middle lobe bronchus, but it appeared probable that the bronchus had been compressed by an adherent node.

Microscopic Microscopic examination confirmed that the discrete nodules were composed of whorls of hyaline material containing crystals which were demonstrated by the polarizing microscope. The middle lobe showed a very marked chronic inflammatory reaction, with dense fibrosis, fetalization of alveoli, and dense accumulations of chronic inflammatory cells. The hilar nodes showed a very marked chronic inflammatory reaction with typical silicotic nodules.

Diagnosis—Chronic bronchiectasis, chronic interstitial pneumonitis, fibrosis of the lung, silicosis of the lung.

Incineration of the lung gave the following result: ash 2.32 per cent of dry weight of lung, silica 0.23 per cent of dry weight, silica 9.7 per cent of ash.

CASE 2—Mr. A. M., aged 69 years, was referred to the Toronto General Hospital on April 2, 1948, with a diagnosis of bronchiogenic carcinoma. The patient had been perfectly well and able to do hard work until six months before admission, when for the first time he developed a cough. This had become frequent and severe, and increasingly more productive until he was bringing up 60 to 90 c.c. of thick dark sputum per day, never foul smelling or frankly purulent, and no blood was seen. Three weeks before admission he developed constant aching pain referred to the right scapula, not affected by breathing or posture. There had been a moderate decrease in exercise tolerance, but no weight loss. The patient had worked as a molder in a foundry for fifty-five years.

On examination the patient was small and wiry, weighing 122 pounds. The trachea was shifted slightly to the right, there were no enlarged lymph nodes in the neck or axillae. Chest expansion was $1\frac{1}{4}$ inches. The right chest posteriorly showed an area of diminished percussion note, decreased breath sounds, and some coarse and medium rales.

Roentgenograms of the chest (Fig. 2) in the posteroanterior view demonstrated a rather diffuse and irregular area of density, which in the lateral view was seen to be a clear-cut density, of a wedge shape, occupying the upper part of the superior segment of the right lower lobe, without evidence of atelectasis. Scattered through the left lung were three small calcified lesions. There was no nodulation of the type seen in silicosis. Bronchoscopic examination showed marked narrowing of the right main bronchus to one third or one fourth of the normal size, just below the origin of the middle lobe bronchus. There was no evidence of tumor. Mucoid secretion was coming from the lower lobe. Multiple sputum examinations were negative for cancer cells.

It was thought that the most probable diagnosis was bronchiogenic carcinoma of the right lower lobe, thoracotomy was done on May 3, 1948. The right chest was opened by resection of the sixth rib. The lung was densely adherent to the chest wall. The superior segment of the lower lobe contained a hard, irregular mass 3 cm. in diameter which extended to the hilus, and on palpation seemed typical of carcinoma. About the hilus were many enlarged lymph nodes, some hard and gray, others large and black. One of the former was removed, and on frozen section showed only inflammatory reaction. Pneumonectomy was undertaken, but the nodes were so densely adherent to the hilar structures that the pulmonary artery was torn, necessitating opening the pericardium and ligation of the vessels within the pericardial sac.

The patient made a good recovery from operation, but developed an empyema which healed satisfactorily with drainage and antibiotics. He was discharged from the hospital on June 4, 1948. On Feb. 4, 1951, he reported that his health was satisfactory, that he was unable to work, and was short of breath on slight exertion.

Pathologic Examination—Gross The superior segment of the lower lobe contained a mass which on section proved to be 5 by 4 cm. It was gray in color, with scattered alveolar pigment, giving a mottled effect. The whole lung showed a considerable amount of anthracotic pigment.

Microscopic Microscopic examination of the solid area demonstrated that it consisted of masses of fibrous tissue interspersed with fibrotic nodules showing a typical lamellar structure. The alveolar septa were thickened due to increased fibrous tissue and the presence of macrophages. The alveoli were mainly collapsed and contained pigment bearing macrophages. The lymph nodes showed a similar fibrous tissue reaction.

Diagnosis—Chronic interstitial pneumonitis (localized) and silicosis.

A portion of the lung was ashed with the following results: Lung ash 25 per cent of dried weight total which 3.75 per cent of dried weight total silica 45.6 per cent of the lung ash.



Fig. 1. (A) and (B) Microscopic examination of the lung tissue showing the presence of anthracotic pigment and alveolar septa thickened due to increased fibrous tissue and the presence of macrophages. The lymph nodes showed a similar fibrous tissue reaction.

middle lobes, the shadow of the left hilus was increased, but there was no evidence of silicotic nodulation. Bronchoscopic examination showed a general mild inflammatory reaction of the right bronchial tree with no evidence of tumor. Laminograms showed a very dense mass surrounding and narrowing the right upper lobe bronchus, with the characteristic appearance of a bronchiogenic carcinoma. Multiple sputum examinations were negative for malignant cells.

The provisional diagnosis was bronchiogenic carcinoma of dubious operability.

Right thoractomy was performed on Feb 15, 1949. The upper and middle lobes were densely adherent to the parietal pleura. A hard irregular mass appeared to arise at the upper lobe bronchus and fill the central portion of the upper lobe and to extend across an incomplete fissure into the middle lobe. Throughout the remaining portions of the lung were scattered many small discrete densities, up to 0.5 cm in diameter. In the hilus were many very firm, gray, adherent nodes up to 1.5 cm in diameter. One was removed, examined by frozen section, and reported to show "old tuberculosis and anthracosis, no evidence of tumor." A dissection pneumonectomy was done, this was difficult because of the nodes which were densely adherent to the great vessels and bronchus.

The patient made a good recovery and was discharged eighteen days after operation. He has been seen regularly since, the last time in September, 1951, and has remained well, with no cough, sputum, or hemoptysis, has gained 30 pounds, and has fair exercise tolerance.

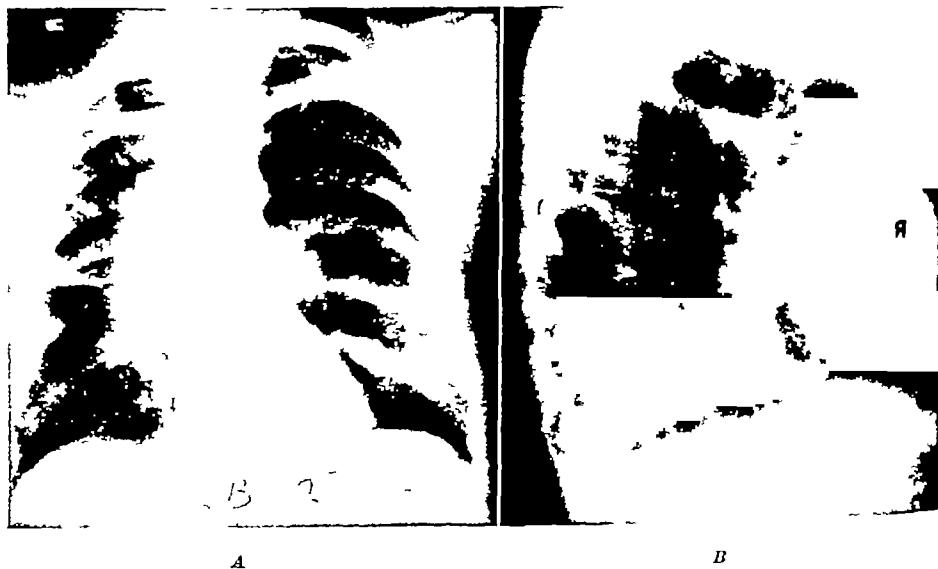


Fig 3—Case 3. A and B. Massive density involving right upper and middle lobes.

Pathologic Examination—Gross. Section of the lung confirmed the presence of a large irregular mass 8 cm in diameter in the upper lobe, extending into the middle lobe. The cut surface was light gray mottled with black. This mass appeared to compress the origin of the upper lobe bronchus, but the more distal portions of this bronchus were dilated and contained mucous casts. The lymph nodes were very hard and enlarged up to 2.5 cm in diameter, some were light gray on section, flecked with black, others were softer and jet black.

Microscopic. Microscopic examination showed that the dense areas consisted of a heavy deposit of fibrous tissue, with an infiltration of chronic inflammatory cells, mixed with nodules composed of concentric layers of hyaline material. Many doubly refractile crystals were present. The upper lobe bronchus showed typical bronchiectasis. The lymph nodes showed dense fibrosis with silicotic nodules.

Diagnosis—Tuberculosis of lung and lymph nodes, chronic interstitial pneumonia and pleural effusion of the upper lobe.

CASE 4—Mr. J. C., aged 57 years, was admitted to the Toronto General Hospital on July 19, 1951. He had no complaints and was referred because of a shadow in the right lung covered on routine radiograph. He had had regular examinations of his chest since 1949, and in 1949 a shadow was noticed radiating out from the right hilus (Fig. 4). Examination at another hospital in January 1950, included bronchoscopy. No evidence of carcinoma was found, and no definite diagnosis was made. Further films (Fig. 4 b) were used to indicate an extension of the shadow, and he was referred for reinvestigation. The patient had no cough, no history of hemoptysis, no chest pain, no weight loss, and good general tolerance. He had worked in a stove and furnace foundry for thirty-five years. He had had some exposure to dust. He looked perfectly well. The only abnormal finding, was slightly decreased posterior costate at the right base posteriorly.



Fig. 4

Fig. 4 a—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 b—Lateral view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 c—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 d—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 e—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 f—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 g—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 h—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 i—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 j—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 k—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 l—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 m—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 n—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 o—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 p—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 q—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 r—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 s—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 t—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 u—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 v—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 w—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 x—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 y—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe. Fig. 4 z—Posterior-anterior view in 1951 showing a shadow in the right hilus with indeterminate lateral extension. Lateral view demonstrates that it is in superior segment of lower lobe.

sible, it was decided not to undertake thoracotomy. His physician was advised to continue to keep the lesion under observation by repeated radiographs and to have further sputum cultures done.

CASE 5—Mr Wm V A, aged 58 years, was referred to the Toronto General Hospital on Dec 19, 1951, with a provisional diagnosis of bronchiogenic carcinoma. He had been in good health until October, 1951, when he developed pneumonia. With treatment by antibiotics he made a slow recovery, but continued to have a cough with a small amount of mucopurulent sputum, lost 12 pounds in weight, and tired easily. There was moderate shortness of breath on exertion. During the pneumonia his sputum was streaked with blood. He was referred because of a persisting density in the left lower lobe on radiographic examination. For many years he worked in smelters and foundries, and had had much exposure to dust.



Fig 5—Case 5 A and B Diffuse density in superior segment of left lower lobe

The patient looked older than the stated age, but was in good general condition. There was a small area of dullness with diminished breath sounds below the angle of the left scapula. Roentgenograms (Fig 5) showed a diffuse area of density in the left lung field, which in the lateral film was shown to occupy the upper part of the superior segment of the lower lobe. A lipiodol bronchogram showed failure of filling of the left superior segmental bronchus. A bronchoscopic examination showed some narrowing and distortion of the left lower lobe bronchus without evidence of tumor. Aspirated secretions and three specimens of sputum were negative for malignant cells. Vital capacity was 3,500 cc, or 80 per cent of the calculated normal.

In view of the long history of exposure to dust, and the lack of evidence of carcinoma on bronchoscopic and sputum examination, it was considered that the most probable diagnosis was obstructive pneumonitis secondary to bronchial obstruction by silicotic nodes. It was decided to do a thoracotomy to rule out carcinoma, and in the hope that the condition could be treated by lobectomy.

On Dec 26, 1951, a left thoracotomy was done. The whole left lung showed a heavy deposition of anthracotic pigment, and a few scattered hard nodules 3 or 4 mm in diameter could be palpated. The upper lobe was voluminous, and the lower slightly reduced.

in volume. The superior segment was filled by an irregular firm mass, and was densely adherent to the parietal pleura. There was moderate thickening about the hilus, with many hard black adherent nodes, up to 1.5 cm in diameter. A dissection lobectomy of the lower lobe was accomplished with difficulty and in doing so it was necessary to free a collar of the e nodes which surrounded and compressed the superior segmental bronchus at its origin. The lobe was immediately examined by frozen section, and no evidence of carcinoma was found.

The patient recovered well, and was discharged from the hospital after eleven days. At that time he was free from cough.

Pathologic Examination—*Gross* Section of the lung showed that in addition to the collar of hard nodes surrounding the superior segmental bronchus, there was a similar hard black node adherent to the bifurcation into the superior and inferior divisions of this bronchus, distorting and narrowing both of the divisions. In the superior segment was an irregular firm mass, gray in color, flecked with black areas, and becoming necrotic in the center.

Microscopic Microscopic examination revealed that there was widespread necrosis in the firm area noted in the gross. The immediately adjacent lung showed a marked fibrous tissue thickening of the alveolar walls, infiltration by acute and chronic inflammatory cells, and many macrophages lying within the alveoli. The lymph nodes showed a heavy deposit of anthracotic pigment, fibrosis, and silicotic nodule.

Diagnosis—Pulmonary abscess, diffuse interstitial fibrosis, and anthracosilicosis of lymph node.



Fig. 1. Diffuse nodulation typical of silicosis, triangular, baylow at right base and disk defects of right lower lobe and hilum. Density in right mid lung field.

Course—Mr. L. S., aged 49 years, was referred to Dr. N. S. Shenstone on Nov. 23, 1945. He was a grinder and chipper in a foundry for more than twenty years. In 1944 a routine chest x-ray showed early diffuse nodulation indicating silicosis, and he was advised to stop occupation which he did not do. For several years he had been losing weight and had a cough which was otherwise well until January, 1945, when he developed a acute febrile illness which was diagnosed as lobar pneumonia affecting the right lower lobe. He gradually recovered but continued to have cough and sputum occasionally blood-tinged and the chest x-ray continued to show nodulation. This led to a further chest x-ray (Fig. 2) which showed diffuse nodulation typical of early silicosis. At this time there was a shift

of the mediastinum to the right, a triangular density at the right base indicating atelectasis of the lower lobe, and an irregular area of density extending upward from the level of the hilus into the upper lobe area.

When referred nine months later there was no significant change in the radiographic picture. A right bronchogram confirmed the presence of an obstruction near the origin of the lower lobe bronchus, with atelectasis of the lower lobe, and a deformity of the middle-lobe bronchus apparently due to a mass of lymph nodes at the hilus. A bronchoscopic examination showed a stenosis of the bronchus to the lower lobe without evidence of tumor. The bronchus contained bloody secretion. Sputum examinations were negative for the tubercle bacillus on smear.

It was concluded that the patient had pulmonary silicosis, complicated by bronchiogenic carcinoma, or possibly by tuberculosis, and thoracotomy was advised.

At operation on Feb. 12, 1946, by Dr. Shenstone, it was found that there were only a few pleural adhesions. The right lower lobe was atelectatic and extremely hard, continuous with it was a hard mass occupying the hilus of the lung and extending into the posterior part of the upper lobe. The overdilated middle lobe and anterior portion of the upper lobe contained many hard nodules like small peas. A dissection pneumonectomy was done.

The patient made a good recovery from the operation and was discharged from the hospital eleven days later. It is reported by his doctor that he remains well, but is short of breath on exertion. Roentgenograms at frequent intervals have shown no significant change in the silicotic reaction in the left lung.

Pathologic Examination—Gross. Extending from the hilus into the posterior segment of the upper lobe and filling the atelectatic lower lobe was a hard mass, blue black on section. This mass surrounded the bronchi, reducing them to slits. In the lower lobe was an abscess 1.5 cm. in diameter.

Microscopic. Sections from the small discrete nodules and from the main mass showed a typical silicotic reaction, with dense areas of hyaline material. The center of some of the nodules showed necrosis. The abscess cavity showed caseation with destruction of part of a bronchial wall. In some areas the bronchi were compressed by a dense silicotic fibrosis, and in other areas they were dilated and showed tuberculous ulceration.

Diagnosis—Silicosis and secondary tuberculosis.

CASE 7—Mr. S. S., aged 63 years, was admitted to the Toronto General Hospital on Oct. 25, 1948. This retired postman had been well until July, 1948, when he began to notice increasing fatigue and weakness. In September he began to cough and raise a few cubic centimeters of yellow sputum, but no sign of blood. About the same time he noticed a huskiness of the voice and a desire to clear his throat repeatedly. He developed a mild ache in the right anterior chest, and noticed increasing shortness of breath on exertion. He had lost 15 pounds since July. The patient had been a postman for thirty-eight years. It was only after evidence of silicosis had been found at operation that direct questioning revealed the fact that he had been a driller in the silver mines at Cobalt for two years before entering the postal service.

He did not appear ill. The trachea was deviated to the right, and there was dullness with diminished air entry over the right chest anteriorly. The vocal cords moved normally. Roentgenograms (Fig. 7) demonstrated a wedge-shaped density occupying the anterior segment of the right upper lobe. Laminograms showed large rounded densities in the right hilus and under the carina suggestive of enlarged nodes. There was no evidence of silicosis. Bronchoscopic examination showed widening and fixation of the carina. Purulent material issued from the right upper lobe bronchus, the middle lobe bronchus was patent, but just below this there was a uniform stenosis of the lower lobe bronchus to 4 mm. Sputum and aspirated secretions were negative for malignant cells and for tubercle bacilli on smear.

Bronchiogenic carcinoma was considered the most likely diagnosis, although the demonstration of many markedly enlarged nodes about the hilus suggested the possibility of obstructive pneumonitis due to bronchial compression by a node.

At operation on Nov. 2, 1948, the right chest was opened. The upper lobe was adherent by vascular adhesions. The anterior segment of the upper lobe was occupied by a very hard

ness which ran into the hilus. The whole bronchovascular tree running down into the lower lobe was markedly thickened, stiff, and irregular. Many enlarged nodes surrounded the hilus and were densely adherent, some were smaller, gray, and hard, others were large, soft, and jet black. Under the carina was a node as large as a big chestnut, black, and soft. These findings were considered typical of a silicotic reaction in the nodes, but one could not be certain that there was not a carcinoma also. Lobectomy was impossible because of the exceedingly dense reaction about the vessels. A dissection pneumonectomy was done with some difficulty.

The patient made a good recovery and was discharged ten days after operation. He has remained well, has regained his normal weight, has a fair exercise tolerance, and does light work.

Pathologic Examination—Gross. Cutting the lung showed that the very hard consolidation of the anterior segment of the upper lobe was a dark, mottled, slate gray in color. There were similar smaller areas scattered through the upper and lower lobes, which were both deeply pigmented, but the middle lobe was crepitant and pink in color. Dissection of the upper lobe bronchus showed that a hard, black node 1.5 cm. in diameter was densely adherent to the anterior segmental bronchus, compressing it and eroding the wall. Other similar nodes were adherent to the lower lobe and middle lobe bronchi.



Fig. 1.—Chest X-ray in *A* and *B*. Marked deviation of trachea to right and density indicating consolidation of anterior segment of right upper lobe. Laminogram shows the marked reaction of the upper lobe bronchus and the shadow of large lymph nodes at the hilus.

Microscopic examination of the area of consolidation showed that it consisted of dense masses of alveoli with many scattered hyaline nodules. There was an infiltration with chronic inflammatory cells, and the alveoli were compressed and filled with cellular and macrophages, some of which contained areas of necrosis. The lymphatic channels had no laminated fibrous reaction with heavy deposits of carbon pigment. There was no evidence of tuberculous infection.

Discussion—Tuberculosilicosis

Case 1.—Mr. B. C., age 54 years, was admitted to the Toronto General Hospital on May 1, 1941. His first chest symptom was a dry, irritating cough in the autumn of 1939 which was followed by a cold. He had fever and was treated for pneumonia. In the spring of 1940 he had a dry cough, loss of weight, and a persistent fever. A chest X-ray showed a large consolidation in the left upper lobe. A biopsy of the consolidation

that it had cleared. He remained perfectly well, with no cough or sputum, until December, 1950, when he had a similar febrile attack and spent ten days in bed. He again recovered completely and became free of cough. In April, 1951, a routine chest film showed an area of density in the left upper lobe and he stopped work. During the month before admission, while he was not working, he gained 12 pounds. For several years he had noticed moderate shortness of breath on exertion. This man had been a miner since the age of 16 years. From 1920 until 1930 he worked underground in a hard rock gold mine where the incidence of silicosis is high.

Examination showed a large, powerful man weighing 230 pounds. The only significant finding was slight reduction in air entry into the left upper lobe posteriorly. Roentgenograms (Fig 8) demonstrated a wedge shaped area of density occupying the apicoposterior segment of the left upper lobe. A laminogram showed a very dense area in the extreme apex, and below this a large triangular shaped density with a very clearly defined margin. The hilar shadows on both sides were greatly increased in bulk and density, but there was no diffuse nodulation. A bronchoscopic examination showed edema and reddening about the orifice of the left upper lobe bronchus. No biopsy was obtained but secretions were aspirated. This specimen and three specimens of sputum were negative for malignant cells. No acid fast bacilli were seen on smear.



Fig 8—Case 8. A and B. Diffuse density at the left apex and marked increase in bulk and density of right hilar shadow. Laminogram shows a very dense shadow at the apex with a less dense triangular shadow below.

In view of this patient's occupational history and the other evidence, it was thought that the density represented a conglomerate silicotic lesion, but carcinoma could not be excluded. It was decided to do a thoracotomy and, if the diagnosis was confirmed and carcinoma could be ruled out, to perform a lobectomy. If this was not possible for technical reasons, it would not be desirable to do a pneumonectomy, because the disability did not justify the severe impairment of respiratory function which would result in this very big man whose vital capacity was 2,902 cc or 55 per cent of the calculated normal.

At operation on June 1, 1951, the left chest was opened. The apicoposterior segment of the upper lobe was filled by an extremely hard mass and was very densely adherent to the parietal pleura. The lobe was freed. Scattered through the lower lobe and anterior part of the upper lobe were hard nodules 2 to 3 mm. in diameter, one was excised. On section it was jet black, frozen section showed a typical silicotic lesion. About the hilus of the lung

there was a dense fibrosis and many gray stony hard nodes, up to 1.5 cm in diameter, were present. The bronchovascular tree to both upper and lower lobes was greatly thickened, irregular and stiff. It was decided that a dissection lobectomy was impossible, but a pneumotomy could be done. The main density was cut into and a generous biopsy was removed. The cut surface was black. Frozen section showed silicosis. The chest was closed.

Further study of the sections confirmed the diagnosis of silicosis with no evidence of tuberculosis. The polarizing microscope revealed many doubly refractile crystals.

The patient recovered well and was discharged from the hospital twelve days later. One month after his operation the patient felt well and was anxious to return to work as a miner, but a report was received that a sputum culture planted two months previously had grown a typical tubercle bacillus. In August, 1951, he had a recurrence of pneumonia and further sputum cultures were done, of which three were positive for tubercle bacillus. He was admitted to the Toronto Hospital for Tuberculosis on Sept. 6, 1951.

This patient undoubtedly has tuberculosilicosis and illustrates the difficulty of recognizing the presence of tuberculosis in a massive density even when biopsy is done. It is probable that he will require resection to control his tuberculosis.

SUMMARY OF CASES

The table summarizes the symptoms of which these patients complained. The oldest patient was 69 years of age and the two youngest were 49, with an average age of 58 years. Five had had prolonged exposure to dust, three as foundry workers, one as a grinder, and one as a miner. Of the other three, one had worked underground in a mine without direct exposure, one had been a sandblaster for a year, thirty-five years previously, and one had been a driller in a hard rock mine for two years, thirty-eight years previously.

TABLE I

CASE NO.	AGE	PNEUMONITIS	SPUTUM	HEMOPTYSIS	CHEST PAIN	LOSS OF WEIGHT (LB.)	SHORTNESS OF BREATH
1	49	++	+	-	-	30	++
2	69	0	++	0	+	0	+
3	42	++	+	+	-	21	+
4	57	0	0	0	0	0	0
5	58	+	+	+	0	12	+
6	49	+	-	+	0	15	++
7	63	0	+	0	-	15	++
8	51	++	0	0	0	0	+

The right lung was involved in six patients and the left in two. The lobes and segments involved were as follows: right middle lobe superior segment of right lower lobe; right upper and middle lobes superior segment of right lower lobe; superior segment of left lower lobe; right upper and lower lobes; anterior segment of right upper lobe; and apicoposterior segment of left upper lobe.

Seven of the patients were subjected to thoracotomy and in five a dissection lobectomy was done; in one a dissection lobectomy, and in one biopsies taken. A thorough examination of the excised lung has failed to show evidence of tuberculosis in four patients. In two others the lung showed definite silicosis. The patient who had biopsies only failed to show tuberculosis, but his sputum was positive on culture; he was considered to have tuberculosilicosis. In the patient who has not been operated upon the

diagnosis has been made on clinical grounds, and, since sputum cultures are negative for the tubercle bacillus and the lesion appears stationary, he is considered to have silicosis

DISCUSSION

The picture found at thoracotomy in this group of patients is characteristic, sufficiently so that in the later cases it was easily recognized even when a history of exposure to dust had not been obtained and there was little reason to question the preoperative diagnosis of bronchiogenic carcinoma. The gross appearance of conglomerate silicosis and tuberculosilicosis may be identical, the distinction can be made only by careful microscopic study. Typically, the massive density has a segmental distribution, and this is fairly accurately maintained. The involved segment may retain its normal volume or be somewhat reduced, but in only one case was there complete atelectasis. The area is usually adherent to the parietal pleura by adhesions which are thick and avascular. The main mass is slightly irregular and stony hard, not rubbery. If it is cut into, the surface is black to dark gray, with a mottled or marbled appearance. The rest of the lung is usually rather voluminous, shows more anthracotic pigment than usual, and on palpation one can feel a few or many hard nodules, usually three or four millimeters in diameter. The lung root shows a characteristic type of chronic inflammatory reaction with many enlarged lymph nodes firmly bound to the vessels and bronchi by very dense fibrous tissue. Palpation reveals that the whole bronchovascular tree, including that to the uninvolved lobe, is thickened, irregular, and stiff, it resembles the gnarled and twisted limb of an old tree. The enlarged lymph nodes vary from one to two centimeters in diameter and are of two types, both commonly seen in the same patient. Some are very hard, and light gray externally, when cut they show white or light gray areas mottled with anthracotic pigment. Other nodes are smaller, softer, rupture easily, and are jet black.

Gross examination of the lung after removal has confirmed the segmental distribution of the massive density. Dissection of the bronchus to the involved segment has shown in most cases that a node is compressing and narrowing it but not obstructing it completely. In one case a hard node was actually eroding the bronchial wall. The bronchi in the involved segment are deformed, showing dilatation in some areas, and in others they are compressed by the surrounding dense tissue, the mucosa is inflamed, and in some cases has shown ulceration.

Microscopic examination demonstrates that the massive densities from different cases, or even different areas in the same specimen, show varying degrees of a chronic inflammatory reaction. In some there is thickening of alveolar walls with marked reduction in the size of the alveoli, and in others this has progressed to complete disappearance of alveoli and replacement by masses of fibrous tissue interspersed with groups of pigment containing macrophages. Scattered through all areas is a sprinkling of inflammatory cells, which tends to be more acute in the earlier stages, with focal areas of suppuration, and of a more chronic type in the later stages of the reaction. The fibrosis is particularly

marked about the bronchovascular bundles, the bronchi are compressed, the vessels show a definite sclerosis, and the lymphatic channels are obliterated or choked by macrophages filled with black pigment. Mixed with the fibrotic areas are masses of laminated hyaline material, characteristic of a silicotic reaction. The small firm nodules scattered through the lung are typical silicotic

Fig. 9



Fig. 10

Fig. 10. A bronchovascular area showing the dense fibrous reaction, a variety of cells, and clear cells of the vessels. The lymphatic and venous channels are thickened by cellular infiltration and are filled with inflammatory cells. (H&E, 100X)

Fig. 11. Tubercle formation. A large, irregular, dense nodule of the lung, containing a large number of small, dark-staining cells, typical of a tubercle. (H&E, 100X)

nodules The hard gray lymph nodes are made up largely of masses of hyaline material, and mature fibrous tissue, the black nodes show silicotic nodules, fibrous tissue, and masses of macrophages filled with pigment

The cases of tuberculosilicosis have shown a background picture like that just described, but added to it are more or less extensive areas of caseation, usually grouped about the bronchi. A point of interest is that the nodes which were adherent to and compressing the segmental bronchus showed a silicotic but not a tuberculous reaction.

From a study of the history of these patients, their clinical signs and the succession of changes shown in the serial roentgenograms which were available in some cases, together with the gross and microscopic examination of the excised tissue, it is possible to formulate a theory of the pathogenesis of the massive densities. There has been a growing realization among surgeons and pathologists that chronic bronchial obstruction, be it partial or complete, may lead to a chronic inflammatory process in the affected segment of lung, and that the picture seen varies from case to case depending on the duration and degree of the obstruction, the rate at which it has progressed, and the number of phases of acute or subacute infection which have occurred in the lung parenchyma. If the obstruction has developed quickly, before there has been much infection, atelectasis is a marked feature, if the obstruction progresses slowly and recurring infection has fixed the lung by fibrosis, there is little atelectasis, but bronchial damage is severe, resulting in secondary bronchiectasis. Paulson and Shaw⁸ described this condition as it is seen in the middle lobe, and remarked that chronic atelectasis and pneumonitis may occur in any portion of either lung. They stated that a chronic pneumonitis develops as a result of atelectasis. The author is more inclined to believe that they are both due to the same cause, namely bronchial obstruction, and that the degree of either is variable. McDonald, Harrington, and Clagett⁹ took this view and preferred to use the term obstructive pneumonitis for the condition, this term does seem to sum up better than any other the essential factors in this particular form of lung infection. They recognized clearly that to produce the characteristic changes in lung the bronchial occlusion need not be complete. In our own material we have found examples of chronic pneumonitis of segmental distribution due to bronchial compression by tuberculous nodes, sarcoid, nonspecific lymphadenitis (middle lobe syndrome), and carcinoma. The involved lung in each case has shown a similar type of dense fibrosis with secondary bronchiectasis.

It is suggested that the so-called conglomerate lesions of silicosis are areas of obstructive pneumonitis due to partial or complete bronchial occlusion from involvement of segmental bronchi in a dense fibrous tissue reaction laid down around the lymph nodes, which are themselves the site of a silicotic reaction. Some of the effect may be due to actual mechanical compression of the bronchus, some due to physiologic obstruction by fixation of the bronchial wall, and some of the changes in the lobe or segment may be due to lymphatic obstruction. All the lymph drainage from a lobe of lung must filter through

the lymphatic channels and nodes in the lobar hilus, and in the presence of silicosis the resulting dense fibrosis of both the nodes and the peribronchial region must cause some degree of lymphatic obstruction. The partial bronchial obstruction determines the localization of infection in the affected segment, the chronic lymphatic obstruction with lymphedema prevents resolution of the inflammatory reaction and leads to the fibrosis typical of 'unre-

Fig. 11

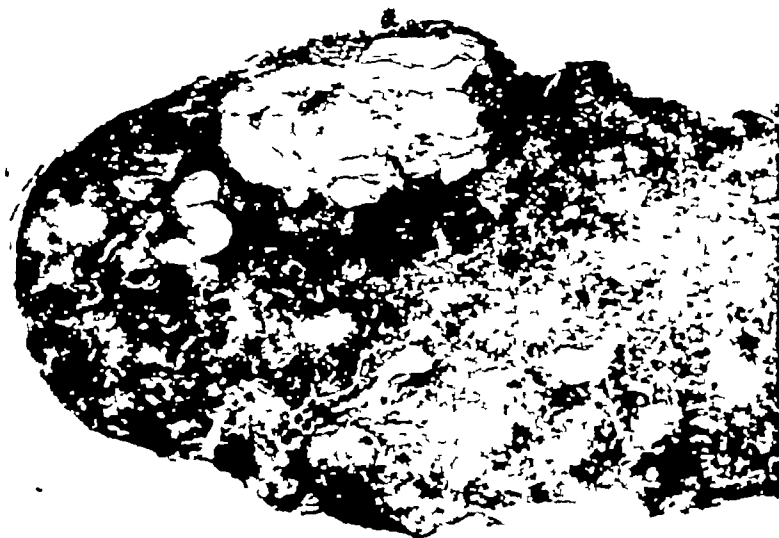


Fig. 12

Fig. 11 (a) - A bronchus at its bifurcation, between two lymph nodes. A little reaction but no evidence of tuberculous. (x55)
 Fig. 12 (a) - A hard black lymph node packed with hyaline material, surrounded by a dense cellular reaction. (x55)
 Fig. 12 (b) - A hard black lymph node packed with hyaline material, surrounded by a dense cellular reaction. (x55)

solved pneumonia" In silicosis, unlike carcinoma, the bronchial obstruction is relatively nonprogressive, so that the end result is a very chronic process in lung—what might be called end-stage obstructive pneumonitis

If this explanation is correct, the importance of the silica as a cause of massive densities is mainly its effect on the lymph nodes This explains some of the anomalies of the occurrence of massive densities, why many patients who have very obvious diffuse nodular shadows never show massive densities, while others who have no recognizable stigmata of silicosis in the roentgenogram may go on to form a massive lesion The massive densities depend on the accident of whether or not a bronchus is compressed by a lymph node The silicotic nodules scattered through the pneumonic area are quite inert and serve chiefly to betray the underlying cause of the reaction The fact that they are more numerous in the dense area than elsewhere in the lung is due to the contraction of the segment pulling them together into a smaller space, and also in those patients who have continued to have exposure to silica after the process has started, to interference with the lymphatic drainage, through which foreign bodies are removed normally

A very interesting question, and one which cannot be answered definitely, relates to the place that tuberculosis plays in this picture Is it an early factor, which perhaps contributes to the lymph node enlargement and the fibrosis, or is it a late invader which finds a favorable medium in an area already fibrotic as the result of recurrent nonspecific infection? A study of these specimens certainly gives the impression that the tuberculosis is superimposed, that the fibrotic background is the same in tuberculosis as in simple conglomerate silicosis In Case 7, in which the anterior segment of the upper lobe showed typical tuberculosilicosis, the segmental bronchus was compressed by a node which was actually invading its wall Sections of this node showed a silicotic, but not a tuberculous reaction, suggesting that the cause for a massive lesion was there before the tuberculosis If tuberculosis is a late invader, this explains why some patients with a massive lesion show positive cultures of sputum only after a long period of observation and many examinations

A point of great interest is the long latent period which may exist between the period of exposure to dust and the development of symptoms and signs of obstructive pneumonitis, in one of our patients exposure had ceased thirty-eight years before and in another thirty-five years previously It is well known that diffuse silicosis of the usual type develops only over a number of years In the case of hard rock gold miners it commonly requires an exposure period of nine or ten years How much of this period is required to receive sufficient dosage of silica to initiate a reaction, and, this threshold dose having been received, how much of the period is required for the development of the specific tissue reaction which constitutes the disease called silicosis, is not known Presumably, the smaller the dosage, the longer the time required to develop a significant reaction It may be that these patients have developed the fibrotic changes in lymph nodes slowly over a period of years and then have for years had some degree of narrowing of a bronchus without serious impairment of function until a respiratory infection has led to edema

of the bronchial mucosa, increased obstruction local pneumonitis, lymphadenitis, further pressure on the bronchus failure of the infection to clear completely, going on to the whole picture of obstructive pneumonitis. In other words these patients may have been vulnerable for years, but only got into difficulties following an infection which initiated a vicious circle. The history of Case 8 strongly suggests this explanation. The patient developed pneumonic attacks with increasing frequency after his first chest infection. He developed an area of pneumonitis in the apicoposterior segment of the left upper lobe which cleared slowly and then recurred in the same area just in the manner which is so commonly seen in bronchiogenic carcinoma.

Even though the picture found at operation in these cases is fairly characteristic it is difficult and at times impossible to be sure in any given case that there is no coexisting carcinoma. There is statistical evidence that there is no relationship between silicosis and bronchiogenic carcinoma. Whether that is true or not, patients who have frank silicosis or like this group of patients have a type of silicotic reaction which is not visible in the roentgenogram, may develop bronchiogenic carcinoma and we have seen examples of both types. In the patient who is known to be a silicotic and may already have massive densities due to silicosis the appearance of another density is likely to be attributed to silicosis until the carcinoma has progressed to the stage of inoperability. When the patient who is not known to be a silicotic, but has the occult form of silicosis described in this paper develops a density, it may be due to chronic obstructive pneumonitis or tuberculosis, or to carcinoma. At operation these conditions are difficult to distinguish even with the help of frozen sections, because in each case there is a firm thickening of a bronchus with consolidation of the affected segment of lung and firm enlarged nodes. If the carcinoma is recognized it may be considered inoperable because of the presence of enlarged nodes which are actually enlarged by a silicotic reaction. If the silicosis is recognized it is then almost impossible to be sure that there is not an associated carcinoma.

The problem of what is the proper treatment for these patients is influenced by the necessity in most cases for a thoracotomy to establish the diagnosis and rule out carcinoma although as pointed out above this is hard to do short of resecting the lung. One of the patients in this group had no symptoms and required no treatment. The other seven were more or less disabled by their disease and in each case the disability was progressive so that treatment was indicated. Because of the structural changes in the lung and the irreversible nature of the process in the lung excision appears to be the only treatment likely to cure these patients permanently or then relapse. The one patient in the group who had thoracotomy without exposure still require excision almost certainly before he can be considered cured of his tuberculosilicosis.

In six of these patients the massive density was confined to a single lobe, four of a lobe and the ideal treatment would have been lobectomy or pneumonectomy. In six of the seven patients subjected to operation the reaction about the bronchovascular tree has been such that

nothing short of a pneumonectomy was possible for technical reasons. In one patient merely lifting the edge of the lung to examine the anterior aspect of the hilus caused an adherent node to pull away from the anterior pulmonary vein tearing a large rent in the vein. In certain cases of tuberculosis with very adherent nodes, in order to accomplish a lobectomy with safety, we have practiced a preliminary isolation and control of the main pulmonary trunk and the two veins, and have then proceeded with the lobectomy and simply repaired any injuries which the vessels sustained. It is possible that this technique might be applied to a silicotic case to permit lobectomy, but in our experience to date even pneumonectomy was difficult. In these patients one probably should not contemplate or attempt resection unless the possibility of pneumonectomy has been considered and accepted.

In assessing the result of treatment it is found that the five patients treated by pneumonectomy are all well in that they have been relieved of their chief complaints, have had no recurrence of chest infection, and have regained their normal weight or more. All are short of breath on exertion, and in three this is fairly severe. One of these three is receiving compensation on the basis of total disability and has become very obese, another is the oldest man in the group and is now 72 years of age, the third is the only one in the group who shows definite evidence of diffuse silicosis in the remaining lung. The remaining two patients who had pneumonectomy are very well and can do light work. It may be pointed out that all these patients complained of shortness of breath before operation. The patient treated by lobectomy is doing well, but insufficient time has elapsed for proper assessment. The patient who had a thoracotomy without resection has shown sputum cultures positive for tubercle bacillus and may require resection. The untreated patient remains well. On reviewing the group it is concluded that the resections which were done were justified.

It is apparent that a question of compensation is involved, and that it is a difficult question because there may be an interval of many years between exposure and the development of disability. A study of the material obtained from these patients strongly suggests that the chain of events leading eventually to severe disability was initiated by the inhalation of silica-containing dust, and would not have taken place if dust had not been inhaled. If that is true, this condition should be considered an industrial disease.

A final consideration is that of a suitable name or description for this condition. It is not silicosis, within the accepted meaning of that term, and may occur in the presence of a very little silicotic reaction in lung. The significant feature is chronic silicotic lymphadenitis, the changes in lung are nonspecific. There is no short way of saying this, perhaps "chronic silicotic lymphadenitis with obstructive pneumonitis (nontuberculous or tuberculous)" is the best that one can do.

SUMMARY

Eight cases of unilateral massive densities in lung due to chronic pneumonitis are presented. The patients were all men, and in each case clinical

and radiographic evidence strongly suggested a diagnosis of bronchiogenic carcinoma

Five patients were treated by pneumonectomy, one by lobectomy, and in another thoracotomy was done and biopsies taken. The eighth patient has not been operated upon.

A study of the excised tissue indicates that the fundamental feature of the pathology in this group of patients is complete or partial bronchial obstruction by pressure of lymph nodes which are the site of a chronic inflammatory reaction secondary to inhalation of silica containing dust. In five patients the chronic obstructive pneumonitis was nonspecific, in three there was an added tuberculous infection.

It is suggested that this condition be called "chronic silicotic lymphadenitis with obstructive pneumonitis (nontuberculous or tuberculous)." "

The patients are all alive, the six treated by excision have been relieved of their complaints.

The author wishes to express his thanks to the members of the staff of the Department of Surgical Pathology of the Toronto General Hospital, and in particular to Dr. W. Anderson and Dr. T. C. Brown for their assistance in the interpretation of the pathology and in the preparation of the microphotographs.

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DISCUSSION

DR. WARRINER WOODRUFF, Saranac Lake, N. Y.—Dr. Kergin has been kind enough to mention the case that Dr. Kelley and I previously reported, and I thought it might be worth while to review that case because it fits in so closely with the description he has

(Slide) This was a 58 year old Polish man who had had thirty years of exposure, ten years in a hard rock coal mine and twenty years in a foundry. This mass was discovered locally when the patient came in for other complaints five years after his last exposure. In all the cases which we reviewed we were struck with the long latent period. There was no case in which no exposure could be proved, but that was proved by chemical analysis. In the case how well outlined this lesion is, and the healing throughout. We missed it at first because of silicosis, we thought it was a malignancy.

(Slide) This is the pneumonectomy specimen and you can see the lesion there. As we reported there were a large number of hard nodules. At the time of operation we suspected the condition. We did not even attempt lobectomy and I now think that it is a fairly acrid, a certain amount of the middle lobe.

(Slide) This is the appearance of the lesion and the area. It shows the diffuse nature of the lesion at the lung.

I would agree with Dr Kergin that the diagnosis may occasionally be made clinically, if it is suspected, particularly in the bilateral case. However, the unilateral case seems to me to pose a more difficult problem, and I am not certain that I would have had the courage not to explore his Case 7 because, as Dr Kergin states, the fact that a patient has silicosis does not mean that he cannot also have a malignancy.

As Dr Kergin points out, exploration in these patients is a more serious problem than in the case of an ordinary tumor because the fibrosis and adhesions at the hilum render lobectomy and segmental resection difficult and hazardous. All seven reported cases of resection have been by pneumonectomy. This is a serious matter because dyspnea is frequently the presenting complaint. Pneumonectomy further increases the dyspnea. For instance, our patient, preoperatively, had a maximum breathing capacity of 84 liters with 30 per cent residual air. Following pneumonectomy and later thoracoplasty, his maximum breathing capacity dropped to 54 liters, while his residual air increased to 41 per cent following resection and 44 per cent following thoracoplasty. Although he survived the operation, he was a "respiratory cripple" and was never able to work.

DR JOHN F HIGGINSON, Portland, Ore—I would merely like to corroborate and confirm some of the points Dr Kergin made with regard to the difficulties of dissection in the hilum in these cases. We have run across only one such case, presenting as a typical right middle lobe syndrome on roentgenologic study, with repeated episodes of infection which necessitated diagnostic thoracotomy. We were able to do a middle lobectomy with difficulty. Stony hard nodes were very intimately bound to the vessels and the bronchus in this area, and we too tore a vessel, the pulmonary artery, where the middle lobe arteries came off. We were able to repair this, fortunately, but I think it should be emphasized that dissection is very difficult. In spite of this, however, with the necessity of making a diagnosis in this type of lesion, I do not feel that a diagnostic pneumonectomy should be done, but rather a lobectomy at the most, although it is very difficult and dangerous in these instances.

DR JAMES M MASON, III, Birmingham, Ala—I wish to discuss Dr Kergin's paper from the medicolegal aspect of silicosis. I am from an industrial section where coal and ore mining are carried on extensively by several large corporations, the United States Steel Company, the Republic Steel Corporation, Woodward Iron Company, the Sloss Sheffield Steel and Iron Company, and many smaller coal companies.

These operators are repeatedly in court defending damage suits covering the disabling effects of silicosis on the coal miners. Anthracosis is common in workers in soft coal. It is very questionable whether silicosis results from working in soft coal, which is the only coal found in this locality.

The coal companies in our section have had the laws so amended that damages from silicosis are settled on the terms of the Workman's Compensation Laws, rather than on the enormous judgments sometimes awarded in damage suits.

DR DAVID H WATERMAN, Knoxville, Tenn—Dr Kergin has given a beautiful picture of this condition in his timely discussion. In the research project we are conducting in Knoxville with the U M W A on silicosis, we are seeing an increasing number of such cases, and during the past two weeks have explored two of them. I wish only to confirm what Dr Kergin has already stated, particularly about the difficulties of segmental or lobar resection. These nodes are like rock, and if you have never had trouble you are in for it now.

DR ALFRED GOLDMAN, Los Angeles, Calif—I want to congratulate Dr Kergin on this excellent report and to emphasize the great importance of this study, because in the past I do not think many of us have felt that silicosis was a surgical condition. But his cases show that certain forms of it are surgical.

I wish to bring to your attention a patient who had not only this syndrome which Dr Kergin has reported, with very large lymph nodes, but also whose disability from infection was continuous over a period of months. In addition to the pathology that Dr Kergin re-

and there was also present, associated with the large nodes, an endobronchial granulomatous exuding process. I wonder if Dr Kergin had any endobronchial disease in his series.

DR. KEPGIN (Closing) —I wish to thank the discussors for their interest in this paper. With regard to Dr Woodruff's comment, I thought it might arouse some doubt in your minds as to why operation was not performed in that patient. Actually, I saw him first at the time of the second x-ray and had I seen him at the time he had the first x-ray I certainly would have operated on him but having had this complete investigation I felt we could temporize in that case. And I was very much influenced by the fact that this man had no symptoms and in my experience I cannot do local surgery in these cases. If I plan to excise the mass I must accept the possibility that it may become necessary to do a pneumonectomy, because of possible accident to a vessel. For this reason I preferred not to operate in this case and I am glad to say he still remains well.

Taking a biopsy does not help very much in excluding carcinoma. The hard mass is similar to the hard mass in the lung with bronchial obstruction due to carcinoma. It simply shows the type of reaction. So I think even biopsies do not exclude carcinoma unless the whole lobe can be removed and examined thoroughly. As Dr Higginson pointed out, a lobectomy can be done in some cases. I think it is much more favorable when the lobe removed is the lower lobe. In the case of upper lobes lobectomy may be technically quite difficult.

Dr Macon has a point that I am not unaware of. This particular subject is so a certain extent dynamite, so far as industry is concerned because if we have a situation where a man is developing disability thirty years after exposure to dust and that disability is due to exposure to dust, then we have a complex and difficult compensation problem. But I think on the other hand that this condition is related to the dust and therefore it is an industrial disease.

With respect to Dr Goldman's question many of these cases have shown bronchiectasis and bronchial ulceration distal to the obstruction. We have not recognized what we could call a granuloma of the bronchus.

NODULAR TUBERCULOSIS

D O SHIELDS, M D (BY INVITATION), JOHN S CHAPMAN, JR, M D,
(BY INVITATION), JAMES CARSWELL, JR, M D, AND
O J WOLLENMAN, M D (BY INVITATION)
DALLAS, TEXAS

DURING the past few years resection of nodular tuberculosis has become rather common. There are a number of reasons for this. Pathologic study of resected tuberculosis as stressed by Medlar has shown that tuberculous nodules usually contain a caseous center often opening into a bronchus. The fibrous wall surrounding nodules may develop slowly or be incomplete. It is thought that the caseous center may later liquify and cavities appear or spreads occur.

Prolonged bed rest usually has little effect on nodular tuberculosis. At times the nodules will decrease slightly in size and become more sharply outlined, but rarely do they disappear except over prolonged periods of time. Antibiotics may hasten clearing of the slight exudation or reversible element of nodular tuberculosis, and in some cases are of value.

Since nodular tuberculosis consists of solid tissue, the relaxation of surrounding lung by collapse therapy will have no effect on resolution of the nodules and will probably not decrease the chances of reactivation.

The above is a simplification of the problem of nodular tuberculosis, but since the point of this paper is a follow-up study of this type of tuberculosis, it will not be gone into more completely.

The development of nodular tuberculosis may occur either by tuberculous pneumonia clearing and leaving nodules, by cavities becoming inspissated, or by the formation of true tuberculomas. Many cases, however, are first seen as nodular tuberculosis, and the method of development is unknown. In addition to the difference in development, the residual nodules may vary from single small nodules to many large confluent masses. It is to be expected that the reactivation rates in these various types might differ. With this in mind a follow-up study of the various types of nodular tuberculosis was undertaken. Figs 1 through 12 show typical examples of each type.

One hundred forty cases were selected from a Veterans Administration Hospital and from private practice for study.

Forty-nine cases were classified as residual nodules left from clearing tuberculous pneumonia. Forty were classified by National Tuberculosis Association standards as inactive at the time of discharge. Two left the hospital before activity could be determined, and seven were active at the time of discharge. Only the patients discharged as inactive need be considered, and, of these forty patients, three cases reactivated.

Thirty-one cases were followed without having previous roentgenograms, so that the development of nodules could not be determined. Twenty-four cases

From the Veterans Administration Hospital McKinney Texas and Southwestern Medical School of the University of Texas Dallas.

Read at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery Dallas Texas May 8-10 1952

were from a Veterans Administration Hospital, and many of these were discovered from routine chest roentgenograms on admission to the hospital. Six of the twenty-four had partial calcification of the nodules, and twenty-one were upper-lobe lesions. Results are shown in Table II.

Fig. 1



Fig. 2

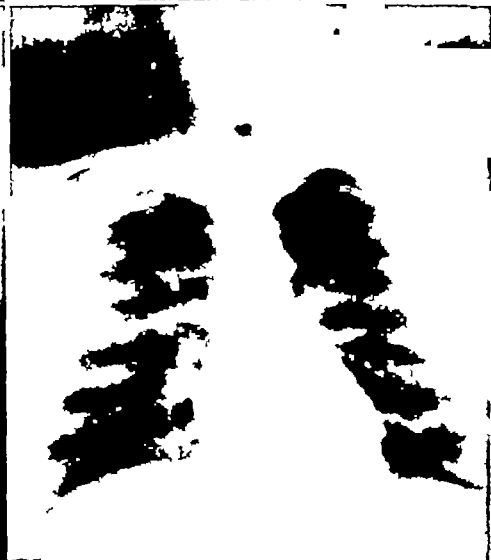
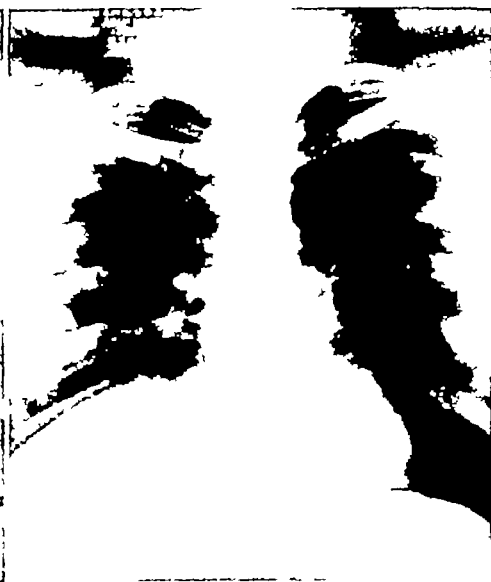


Fig. 3

Fig. 4

- TABLE II
 (A) — Tuberculous nodule in right upper lung field
 (B) — Tuberculous nodule in right upper lung field
 (C) — Tuberculous nodule in right upper lung field
 (D) — Tuberculous nodule in right upper lung field
 (E) — Tuberculous nodule in right upper lung field
 (F) — Tuberculous nodule in right upper lung field
 (G) — Tuberculous nodule in right upper lung field
 (H) — Tuberculous nodule in right upper lung field
 (I) — Tuberculous nodule in right upper lung field
 (J) — Tuberculous nodule in right upper lung field
 (K) — Tuberculous nodule in right upper lung field
 (L) — Tuberculous nodule in right upper lung field
 (M) — Tuberculous nodule in right upper lung field
 (N) — Tuberculous nodule in right upper lung field
 (O) — Tuberculous nodule in right upper lung field
 (P) — Tuberculous nodule in right upper lung field
 (Q) — Tuberculous nodule in right upper lung field
 (R) — Tuberculous nodule in right upper lung field
 (S) — Tuberculous nodule in right upper lung field
 (T) — Tuberculous nodule in right upper lung field
 (U) — Tuberculous nodule in right upper lung field
 (V) — Tuberculous nodule in right upper lung field
 (W) — Tuberculous nodule in right upper lung field
 (X) — Tuberculous nodule in right upper lung field
 (Y) — Tuberculous nodule in right upper lung field
 (Z) — Tuberculous nodule in right upper lung field

TABLE I TUBERCULOUS PNEUMONIA DEVELOPING NODULES FORTY NINE CASES

Inactive (40)	Unstable 7%
Activity undetermined (2)	Unstable 50%
Active (7)	Unstable 13%
Average largest nodule	17 cm
Average time followed	47 mo

TABLE II DEVELOPMENT OF NODULES UNKNOWN THIRTY ONE CASES

Inactive (31)	Unstable 6%
Average largest nodule	15 cm
Average time followed	45 mo

Ten cases of nodules developing from inspissated cavities were found by serial roentgenograms. However, only nine were inactive at the time of discharge from the hospital, and three of these showed reopening of the cavity. Two of these three cases reopened after a period of forty-eight months, and both of these patients had pneumothorax at the time of reopening.

TABLE III INSPISSATED CAVITIES TEN CASES

Inactive (9)	Reopened 33%
Active (1)	Reopened 100%
Average largest nodule	18 cm
Average time followed	49 mo

Only four cases that were considered to be true tuberculomas were found. All of these developed as soft infiltrations which gradually became more sharply outlined but remained the same size. No cases developed from small nodules which became larger as determined by serial roentgenograms. Results are shown in Table IV.

TABLE IV TRUE TUBERCULOMAS FOUR CASES

Inactive (3)	Unstable 0%
Active (1)	Unstable 100%
Average largest nodule	25 cm
Average time followed	29 mo

The last group is made up of cases which vary somewhat in their method of development. Most of the Veterans Administration Hospital patients had initial roentgenograms showing multiple large nodules with little exudation and with insignificant decrease in the size of the nodules with prolonged bed rest with or without streptomycin and para-aminosalicylic acid. Often increase in the extent of the disease occurred merely by new nodules forming. In other words, it was the type of nodular tuberculosis that slowly browses downward through the lungs. However, the cases from private practice contained many with a large element of exudation that cleared with bed rest but left a residual

TABLE V MULTIPLE NODULES WITH FIBROSIS FORTY SIX CASES

Inactive (36)	Unstable 47%
Activity undetermined (1)	Unstable 100%
Active (9)	Unstable 77%
Average largest nodule	15 cm
Average time followed	5 mo

of many nodules with or without a residual or linear fibrosis. Both groups were left with a multiple nodular residual. No cases with bilateral far-advanced tuberculosis in which surgery could not have been considered are included in this or the other groups. Results are shown in Table V.

Fig 5

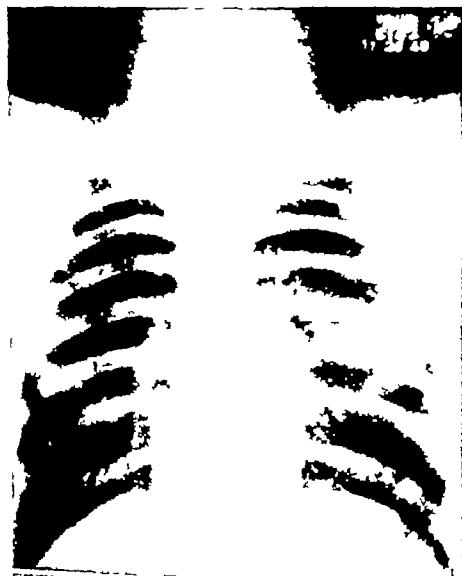


Fig 6

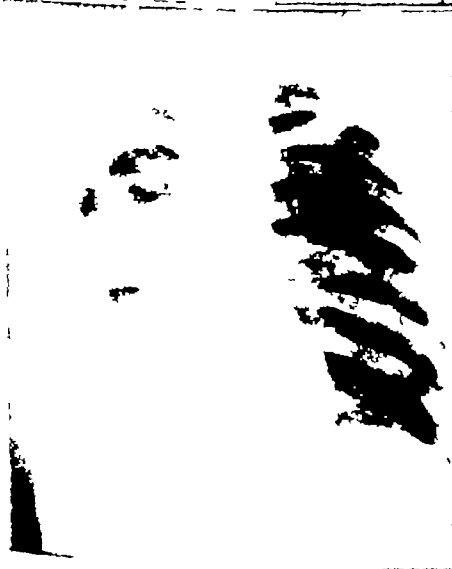


Fig 7

Fig 8

Fig 5 (top). Bilateral cavity with fluid level in the lower lobe. Fig 6 (top). Bilateral cavity with fluid level in the lower lobe. Fig 7 (bottom). Bilateral cavity with fluid level in the lower lobe. Fig 8 (bottom). Bilateral cavity with fluid level in the lower lobe. Multiple nodular opacities in both lungs. Bilateral cavity with fluid level in the lower lobe.

DISCUSSION

With the exception of the small group (4 cases) of "true tuberculomas," all groups were followed for an average period of about four years. No case

Fig 9



Fig 10

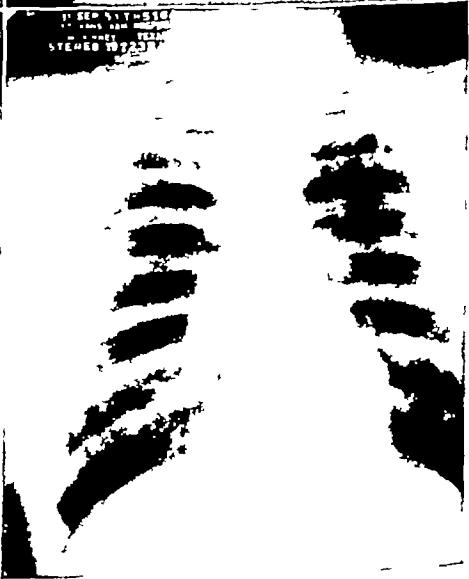


Fig 11

Fig 12

Fig 9 (Case F) —Sharply outlined nodule at left second anterior rib. Hazy infiltrate medial to this in left first interspace.

Fig 10 (Case F) —Soft nodules in left first interspace are more plainly seen than in Fig 9 (Case F).

Fig 11 (Case F) —Nodules sharply outlined as compared to Fig 10 (Case F). This was considered a true tuberculoma.

Fig 12 (Case G) —Multiple nodular tuberculosis left apex. No clearing on two years bed rest. Sputum cultures occasionally positive.

was included in the study if it had not been followed at least six months, and the longest follow-up was one hundred and thirty-nine months. As there is an impression that if a nodular lesion has been stable for a long period of time, it is less likely to reactivate than if it has been stable for a short period of time, an analysis of this was made. Cases followed from six to twenty-seven months were compared to those followed for longer than twenty-seven months. However, no significant difference in percentage of reactivation between the two groups were found. Groups were divided into those of nodules of 1 cm. or less and those with nodules above 1 cm., and again no significant difference in reactivation rate was found.

There were a total of 140 cases in the study and 35 cases (25 per cent) showed reactivation. Of only the 119 cases which were inactive at the time of discharge from the hospital are included, there were 25 cases (21 per cent) that reactivated. A total of 76 cases in the study were from a Veterans Administration Hospital and, of these 76, 18 received collapse therapy. Eleven received a phrenic crush and of these, 4 cases (36 per cent) reactivated. Seven received pneumothorax and 4 cases (57 per cent) reactivated. It is true that only a small group received collapse therapy but we think it supports the view that collapse therapy does not control nodular tuberculosis.

The reactivation rate was high in inspissated cavities and the multiple nodular disease groups but was relatively low in tuberculous nodules of unknown development and cases of tuberculous pneumonia where only a few nodules remained as residual.

TABLE VI. RESULTS OF CASES WITH INCREASE IN TUBERCULOSIS THIRTY FIVE CASES

Resected	16
Inactive on bed rest	2
Inactive pneumothorax	
Thoracoplasty	1
Still active	11 (3 poor prognosis)
Probably active	1
No follow up	1

Of 140 cases studied, 35 cases eventually became unstable either by evidence of positive sputum, increase in the lesion, or reopening of cavity as determined by x-ray study. Four of the patients that developed cavity were treated either by pneumothorax or thoracoplasty and the disease was controlled by collapse. Eleven patients still have active disease and 3 of these have a relatively poor prognosis. However, none of the patients have hopeless disease and none have died. One patient has had no further follow up after evidence of reactivation of his disease. Other results are shown in Table VI.

SURGICAL RESECTION GROUP

Thirty patients who had resection for nodular tuberculosis at least one year previously were selected for follow up study. A total of 36 operations were done on these 30 patients and the type of resection is shown in Table VII.

TABLE VII. EXTENT OF RESECTION

LOBECTOMY	SEGMENTAL	WEDGE OR LOCAL
14	11	11
(13 patients)	(9 patients)	(8 patients)

There were no major surgical complications in this group of patients, and no deaths occurred. The average follow-up was twenty-seven months and 2 patients have shown evidence of reactivation. One patient had a new nodule in the opposite lung two years after resection and one patient had a positive sputum culture without a change in roentgenogram fourteen months following resection. Therefore, 30 patients with nodular tuberculosis (none had cavities) which were followed an average of twenty-seven months showed a reactivation rate of 7 per cent.

TABLE VIII. RESULTS (30 PATIENTS)

SURGICAL COMPLICATIONS	ACTIVITY		AVERAGE DURATION OF FOLLOW UP
	ACTIVE	INACTIVE	
None	2	28	27 mo

Careful pathologic study was done on all surgical specimens. The findings were similar to those described by Medlar, and we do not think it would serve any useful purpose to describe these again.

Since the reactivation rate of the groups, (1) nodules of unknown development, and (2) nodular residuals following tuberculous pneumonia was much lower than that of the groups (3) inspissated cavities and (4) multiple nodules, a comparison of pathology in these various groups was attempted. However, no definite difference as to extent of caseation, thickness of fibrotic wall, and patency of bronchial opening could be determined in these 4 groups of nodular tuberculosis. It has been stated many times in the literature that, because of the pathologic findings in nodular tuberculosis, the lesions are extremely dangerous and the implication is that all nodular tuberculosis should be resected. We think that the pathologic findings in nodular tuberculosis are of great interest, but do not think that they should be the determining factor in selection of cases for resection. We prefer to evaluate the cases individually and resect those in which in our experience the reactivation rate has been high.

Actually, we think that most other groups who are using resection for the treatment of nodular tuberculosis are also selecting cases carefully and are not being influenced by the pathologic findings to a point where all types of nodular disease is routinely being resected.

CONCLUSION

It is dangerous to make generalizations concerning tuberculosis. However, we think the follow-up study of control cases of nodular tuberculosis and evaluation of a small group of resected cases suggest the following conclusions:

1. Inspissated cavities and multiple nodular tuberculosis have a high rate of reactivation.

2 Nodular residuals left from clearing tuberculous pneumonia and nodules of unknown development have a rather low incidence of reactivation

3 Collapse therapy does not prevent the reactivation of nodular tuberculosis

4 Reactivation of tuberculosis following resection of nodular tuberculosis has been low in a small series of cases, suggesting that resection may be a valuable therapeutic procedure in the treatment in this type of tuberculosis

5 We think that resection of nodular tuberculosis should usually be restricted to misspissated cavities and multiple nodular tuberculosis

DISCUSSION

DR JOHN ALEXANDER, Ann Arbor, Mich. This problem is so complex that it cannot be discussed adequately in a few minutes. Without direct reference to Dr Shields' and Dr Warren's series of patients (because sufficient preoperative data have not been presented) but with reference to the present wave of enthusiasm for resection in many cases I have observed a tendency to treat a large proportion of patients with only bed rest and chemotherapy (and without collapse therapy) for a period varying from a few weeks to many months, or even several years. Then, whether the sputum or gastric contents are negative or positive, some form of resection operation is often carried out for the roentgenographic shadows that represent the lesions that have not become absorbed. Although the present mortality rate of resection is satisfactorily low, the morbidity rate is satisfactory and the long term percentage of patients having consistently negative cultures of sputum or gastric contents after the cessation of chemotherapy is even less satisfactory.

Unless and until valid evidence is produced to show that small or relatively small residual roentgenographic shadows (in the presence of consistently negative cultures of pulmonary secretions two or more months after the cessation of chemotherapy) are usually even frequently an important menace to a patient's future health I urge that lesions suitable for collapse therapy according to the standards of a few years ago be treated by collapse therapy and that resection be reserved for those patients who are unsuitable for collapse therapy and in whom it has been used and has failed.

If the frequently expressed assumption is correct that certain shadows represent caseous nodules that are virtually incapable of becoming permanently encapsulated and excluded from the bronchial tree, it is impossible for me to understand why so many thousands of patients who had lesions that had the roentgenographic appearance of small or relatively small caseous foci have lived for many years in excellent health and with negative cultures of pulmonary secretions. My plea is for a far greater discrimination in the selection of patients for resection, and for the use of suitable collapse therapy measures as early in the course of active tuberculous disease.

DR B. J. EVAN, Bay Shore, N. Y. — This paper brings out Dr. McFarland's teaching concerning the residual tuberculous or nontuberculous factor. I believe the recurrence rate which you just presented was from 0 to 15 per cent. If we can cut that rate down from 9 to 15 per cent to the neighborhood of 5 per cent I think we have accomplished something. Dr. McFarland has repeatedly shown that obliterated cavities or filled cavities can communicate with bronchi and contain myriads of tubercle bacilli. The fact that tuberculosis is unpredictable. However, the situation in anterior and inferior segments is likely to give rise to spread of infection in posterior segments. Therefore, if a cavity can be completely obliterated with little morbidity or mortality, it is desirable to do so. It is possible accurately to select the pathologic status of a given lesion by roentgenograms and clinical methods. For example, in a patient with a large cavity I have the culture of pulmonary secretions and sputum.

somewhat analogous to that of subacute bacterial endocarditis. Both can usually be controlled by long term chemotherapy. However, surgical correction of the residual defect is necessary in some cases to prevent recurrence and provide a permanent cure.

DR JOSEPH GORDON, Ray Brook, N Y—I simply would like to tell you about some of the studies we have under way at the Ray Brook State Tuberculosis Hospital. Since we were associated with the original work done at the Sun Mount Veterans Hospital, and recognizing, as Dr Alexander has said, that this is a difficult problem, the role of chemotherapy being applied was not completely understood. We have taken a group of cases under varying courses of chemotherapy and studied them bacteriologically, and found that in a group of these cases treated from three to six months and examined carefully, they too failed to show growth of tubercle bacilli both in the guinea pig and on cultures. We have not been satisfied with this, and have selected a small group of cases with the same criteria used for surgical resection with chemotherapy, that is, a patient with a known case of tuberculosis who has shown no activity radiologically for six months and whose sputum is negative by culture for the same period of time, and have operated without preliminary drug therapy. We have resected these cases and have found that here too we have been unable to grow tubercle bacilli on culture or in the guinea pig. In addition to this, we have under way a study to determine whether or not viable organisms are present after discontinuance of drug therapy for some six months to a year. With no change in the roentgenogram and no evidence of positive sputum, resection in these patients will be done and the material examined similarly for comparison.

We have also been studying some cases by bronchography before and after resection to ascertain what changes take place in the bronchial tree postoperatively, particularly in the wedge type of operation. In some instances angiograms have also been done. In a small group of cases we have found very little evidence of distortion or damage to the lung following the wedge type of resection.

DR FELIX HUGHES, Memphis, Tenn—At Kennedy Veterans Hospital during the six years prior to April 1, 1952, we have performed 178 resections for pulmonary tuberculosis. I would like to make several points, (1) I think we must add a word of caution about too free an application of the principle of the excision of residual tuberculous lesions. I think one could not have a better standard to go by than the presentation of Dr Alexander published in the Transactions of the American Review of Tuberculosis for 1951. To make an adequate choice of the proper treatment of any patient requires extensive knowledge of that patient. Our patients who were operated upon prior to Oct 1, 1951, are reported in our paper presented here by title, and the follow up shows that 18 per cent of these patients that have been resected have had a recurrence of disease. I want to point out, however, that many of these cases were operated upon early in our resection experience for tuberculosis, that we were using only streptomycin, that we were doing many segmental resections, and that we were doing many patients with acute or progressive disease. I think it is very important to point out that these cases must be carefully selected to get good results.

Another point I would like to make is that our results between April, 1950, and April, 1952, are greatly improved over our results prior to that time. Before that time we were using streptomycin alone. Since that time we have used at the time of resection streptomycin and PAS. We have given PAS daily to those patients who can tolerate it. I do not think there is any doubt that the combination of the two gives better results than streptomycin alone. Our results now are beginning to confirm those shown at Sun Mount, that streptomycin given one gram daily with PAS 12 grams daily, for long periods of time, will produce lesions that will show bacilli on smear, but the bacilli will not grow. We have seen this more often after daily streptomycin and PAS than we have seen it with patients who have had streptomycin twice a week and PAS daily. It has occurred in these patients too, but not so frequently.

During the last two years we have done 110 cases with three spreads, three temporary air leaks, and one fistula. All were successfully treated by ward procedures.

Another point about the treatment of late recurrences, the recurrences that occurred after resection were more easily treated than other lesions, and we have obtained better results. I would like to mention resistant cases. I do not think a patient who has bacilli that are resistant to streptomycin should be denied a resection if, from clinical examination, he is a good risk patient. We find that many of these patients have bacilli that are sensitive after the residual cavity with the resistant bacilli has been resected. We have used both the drugs in resistant and sensitive cases, regardless of the sensitivity results. I think resection now is the preferable type of treatment for residual tuberculous lesions in selected cases. The Kennedy Veterans Administration Thoracic Surgery Section staff has now done 255 subtotal pulmonary resections for benign lesions with no deaths. This is in addition to the previously reported Army series of 236 lobectomies with one death.

DR CHAPMAN (Closing). The original purpose of this study was to try to find out what was good to resect and what was good to leave. Our main conclusions run about as follows: nodules that developed from exudative disease, from bronchopneumonia, or from pneumonia disease, regardless of their number or their size, seem on the whole to be relatively benign and not to require resection. Nodules which developed particularly in the apical posterior regions from causes that are unknown—where we have not visualized in part the origin of these nodules, but where we simply know they are nodules from the time they are seen, these nodules are multiple again regardless of their size—have an extremely bad prognosis. That is really not saying anything more than giving in a statistical fashion what everybody has for a long time thought. The type of thing that we are calling nodular disease is the same thing that Dr. Amberson in times past has referred to as indolent tuberculosis which browns downward slowly. That is what we are talking about as representing the type with the highest reactivation rate.

In answer to Dr. Alexander's comment I would simply say that a large number of these patients either had phrenies or pneumothorax or prolonged bed rest, or a number of other things, for possibly two to three years with stability following. And still the patients broke down again. We would regard this type of lesions as ones which fundamentally are not compressible. If they were compressible collapse would seem to be a reasonable procedure. But these are hard; they are shotty and like marbles, and I cannot quite see how a collapse measure which depends largely upon the elastic retraction of the lung itself would have much effect on that kind of firm non-resilient structure.

For that reason, soon after we began to learn from Dr. Medlar what these cases were, we began resecting them. On the whole I think from the figures we have that is about 10 to 20 per cent recurrence rate or disability rate in the original group of nonnodular, with about a 6 per cent reactivation subsequent to resection in this group the management by resection seems preferable. It is still not a good group and I suspect, or hope that in the future as we follow these patients for longer than thirty months, we will have our results.

THE COMPLICATIONS AND THE RESULTS OF TREATMENT OF BRONCHOPLEURAL FISTULA FOLLOWING RESECTION FOR TUBERCULOSIS

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OTTEEN, N C

IN SPITE of the development of chemotherapeutic agents and refinements in surgical technique, postoperative bronchopleural fistula continues to be a frequent complication of resection for tuberculosis

A few studies concerned with the pathogenesis of bronchopleural fistula have been made. Reports dealing with the treatment and subsequent course of a group of such patients, however, are rare. This paper will deal with the complications encountered in a series of thirty patients who developed post resection fistulas between October, 1945, and August, 1950. The methods and the results of treatment will also be discussed. A comparative summary will be made of a group of fifteen patients who developed bronchopleural fistulas following operation and were treated during the past year.

The morbidity rate of pulmonary resection increases markedly whenever a fistula develops. This increase is usually associated with pulmonary or extrapulmonary spread of the disease. Death often results from the effects of the spread. A persistent positive sputum frequently follows the fistula and persistent sinuses are the rule.

Seventeen of the thirty patients with fistulas, or 56.6 per cent, developed pulmonary spreads. One occurred on the sixteenth postoperative day, before the fistula was diagnosed. In four patients a spread developed simultaneously with the fistula. The spreads which occurred concurrently with the fistula were extensive in character. In eight patients a spread occurred after the fistula was diagnosed. These were apparent from two weeks to eight months after establishment of the fistula. The remaining four spreads followed staged collapse procedures or appeared after the fistula was thought to be closed. It should be emphasized that the bronchocutaneous sinus must be kept open until the fistula has definitely closed. If this is not done the air containing pocket around the fistulous opening acts like the bulb of an atomizer and causes a spray of infectious secretions to be forced into the bronchial tree with each violent cough.

The results of pulmonary spreads are shown in Table I. Six patients, or 35.2 per cent, died as a result of the spread. Three, or 17.6 per cent, have progressive disease and in eight, or 46.2 per cent, the disease is stabilized. Four have a persistent fistula.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Read at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery, Dallas, Texas, May 8-10, 1952.

TABLE I RESULTS OF PULMONARY SPREADS

TABLE OF SPREADS	DEATHS		PROGRESSIVE DISEASE		PERSISTENT FISTULA	
	NO	PER CENT	NO	PER CENT	NO	PER CENT
17	6	35.2	3	17.6	4	23.5

Extrapulmonary spreads were less common but were uniformly fatal. Three patients incurred extrapulmonary spreads which contributed to their death. Two of these developed tuberculous meningitis and one tuberculous pericarditis. One meningitis death occurred five months after pneumonectomy and another three and one half months after lobectomy. Tuberculous pericarditis developed in the third patient one month after a parenchymal spread occurred. The patient died ninety days after pneumonectomy.

At the present time six of the thirty patients have persistent bronchointerstitial fistulas in spite of numerous attempts to close them surgically. Two of these have a questionable prognosis and four have shown progressive improvement. A sinus has persisted in seven patients after the apparent closure of the fistula. Six of these have stable pulmonary lesions and a negative sputum. Of the nine patients (Table II) developing a fistula following lobectomy four fistulas, or 44.4 per cent, are closed. Twelve or 57.1 per cent of the postpneumonectomy fistulas are closed.

TABLE II PERCENTAGE OF CLOSURE OF BRONCHOPLEURAL FISTULAS

	NO OF FISTULAS	FISTULA CLOSED	
		NO	PER CENT
Lobectomy	9	4	44.4
Pneumonectomy	21	12	57.1

One pneumonectomy patient died of diabetic coma at home after closure of the fistula.

None of the thirty patients who developed fistulas died. Eight deaths were attributable to complications resulting from the fistula. One patient died from toxemia and cardiac dilatation two weeks after an early postoperative fistula. Three died from the effects of extrapulmonary tuberculosis as previously described. Three died as a result of massive pulmonary spread at one month, four months, and one month after resection. One death from pulmonary embolism occurred twenty months after pneumonectomy. The final death was due to diabetic coma after discharge from the hospital. A fistula which had been closed after pneumonectomy had been closed by means of a thoracoplasty prior to death.

Seven of the nine patients who succumbed were treated with streptomycin. Eight had in addition an open thoracotomy and two in obliterative thoracoplasty. One patient died the day of the spread before treatment could be instituted.

Only one of the thirty patients (Table III) was alive in the year after operation. The others were hospitalized but two of these were making rehabilitation. Four of the hospitalized patients had open thoracotomy and persistent cutaneous sinuses. Ten patients had been discharged

two with open fistulas and three with small chest wall sinuses. Three patients of the series had active disease and a positive sputum. Two of these had a doubtful short-term prognosis.

TABLE III FINAL RESULTS OF TREATMENT

	NO OF FISTULAS	PATIENTS LIVING	PATIENTS DEAD	IN HOSPITAL	DIS CHARGED	FISTULA CLOSED*	PER SISTENT SINUS
Lobectomy	9	6	3	3	3	4	1
Pneumonectomy	21	15	6	8	7	12	6
Total	30	21	9	11	10	16	7

*Seven patients with persistent sinus are included in the closed fistula group

Early diagnosis is of paramount importance in the treatment of broncho pleural fistula. The diagnosis was made an average of fifty-eight days following surgery in this group. Various adjuncts were used in suspected cases to help establish the diagnosis. These included bronchoscopy, instillation of methylene blue, ether, and radiopaque oils into the pleural cavity and the determination of intrapleural pressures. Gas analysis of intrapleural air was tried in two patients but did not contribute to the diagnosis.

TREATMENT

When a diagnosis of fistula was made, treatment was considered to be an emergency matter. The objectives were threefold: first, drainage of the pleural space, second, eradication of the space by the re-expansion of remaining lung tissue or obliteration by thoracoplasty, third, closure of the fistula itself.

In the majority of these patients drainage was established by means of open thoracotomy. This immediately lessened the chance of spread to uninvolved areas. As soon as the patient's condition permitted, obliteration of the potential empyema space by means of thoracoplasty was attempted. An anterior stage under local anesthesia was usually possible within a week or two after drainage. The posterior stages were then completed under local or epidural anesthesia as rapidly as the patient's condition permitted. As the infected pleural space diminished in size the patient's condition generally improved. Frequent blood transfusions were necessary during this period. An average of five or six operative procedures, including partial scapulectomies and Schede type thoracoplasties were necessary before obliteration was attained. When the space had been reduced to a narrow sinus, there were several rather dubious choices to select from. The tracts were delineated with radiopaque solutions and a determination made as to whether to attempt a plastic closure using a muscle graft, attack the bronchial defect directly, or be satisfied with a living patient who had a small draining sinus and an infrequently positive sputum. Our results with plastic closure and muscle grafts have not been encouraging. In a few patients there was apparent early success but the majority subsequently broke down and required further surgery or were treated with long-term chemotherapy.

During the past year our initial attack has been more conservative. As soon as a fistula is diagnosed a large catheter is attached to a Stedman pump and inserted through an intercostal space. This takes care of the immediate emergency and gives time to determine whether the air leak is due to a breakdown of the bronchial closure or to escape from a raw surface left as a result of segmental resection or lobar separation. Fifteen patients (Table IV) have been treated in this way. Four of these were treated with closed suction only. In three the lung was re-expanded and the temporary air leak closed in twenty-four hours. One has a definite fistula and remains on closed drainage. A secondary open thoracotomy was done on the fifth patient and a small empyema space unroofed.

TABLE IV. RESULTS IN FIFTEEN PATIENTS TREATED WITH CLOSED DRAINAGE INITIALLY

	NO.	FISTULA CLOSED	SPUTUM NEGATIVE	DEATHS
Closed drainage only	4	3	3	0
And added thoracotomy	1	0	0	0
And added thoracoplasty	5	5	5	0
And excision of remaining lobe	3	3	3	0
And secondary fistula closure	2	0	0	2
Total	15	11	11	2

Closed drainage followed by a limited thoracoplasty was done on five patients. The fistula closed, the sputum remained negative and the wound healed in each patient.

Early open thoracotomy and excision of a remaining lobe or segment or lobe were done in three patients with complete success. The wounds healed and the sputum became negative. Early open thoracotomy and suture of the fistula were done in two patients. Both fistulas reopened and both patients died.

The results obtained in this group of fifteen patients indicate that closed drainage followed if necessary by a thoracoplasty or secondary resection of a remaining lobe is a desirable method of treatment for postoperative fistula. The mortality rate in this group was 16.6 per cent. Thirteen patients are alive; eleven of them have a negative sputum and a healed wound. Two patients recently operated upon have a small residual fistula and a negative sputum.

Streptomycin and para-aminosalicylic acid were used for long periods in all of these patients with the exception of the one who died before treatment could be instituted. Conservative collapse measures such as pneumothorax were used whenever spreads occurred.

SUMMARY

1. A mortality rate of 30 per cent was encountered in a group of thirty patients who developed a bronchopleural fistula after resection for pulmonary tuberculosis between October 1945 and August 1950. Death was usually due to effects of pulmonary or extrapulmonary spread of the disease.

2 The mortality rate decreased to 16.6 per cent in a smaller group of fifteen patients who were treated by a modified technique during the past year. The period of observation in the later group, of course, is comparatively short.

3 Emphasis should be placed on efforts to avoid bronchopleural fistula by means of careful case selection and preoperative preparation.

4. Chemotherapeutic agents should be administered continuously for a long enough period before operation to stabilize the lesion.

DISCUSSION

DR C. THOMAS READ, Phoenix, Ariz.—There is a period in which the patient is depleted following a fistula, especially postpneumonectomy, and a series of operations is usually necessary. I have had two experiences recently with what I would say were just fair risk patients. One had a fistula ninety days following pneumonectomy for carcinoma, and this man was a respiratory problem at that time. The other patient was a 40 year old man who had a resection for extensive suppurative disease on the right side. He had a fistula develop in thirty days. As soon as the fistula developed, the patient was removed to the hospital from a convalescent center, a tube was inserted into the chest through an intercostal space, and the next morning each patient had a complete thoracoplasty with thoracotomy and repair of the fistula.

(Slide) This is a 40 year old white man who had extensive suppurative disease on the right side. I was forced to do a pneumonectomy in this situation for numerous practical reasons. You will notice on the left side the site of a thoracotomy, at the age of about 8 years, this patient had an empyema. In addition to having had drainage, a previous bronchogram disclosed basilar segmental disease on the left side. I mention this to show that this patient was only a fair risk from the standpoint of pulmonary function. In other words, he had only a partial left lung with which to breathe. This patient tolerated the procedure very well. The thoracoplasty consisted of ribs two through eight, and lysis of the pleura superiorly from beneath the first rib. He had a satisfactory postoperative recovery. The older man, who was 55 years of age, was a much worse risk than this patient from a pulmonary function standpoint, and he recovered in a like manner. Following thoracoplasty tube drainage was accomplished with water seal and suction. Both patients left the hospital in about seven days.

I realize that these operations were done on the right side, and were not done for tuberculosis. However, such a method of management might be feasible in the problem under discussion.

DR DANIEL A. MULVIHILL, New York—I am sure we are all very appreciative of this fine presentation of Dr. Murphy and his associates, and I wish to commend them for their very careful clinical observations, and their critical analysis of their work, which is bound to be helpful to all of us who still have to contend with this distressing complication of resection surgery.

I thought it might be of interest to you to present a parallel experience in a sister institution in the Veterans Administration, and I speak for the group at the Veterans Administration Hospital at Castle Point, N. Y. (600 beds), where Dr. Charles Lester and myself are co-consultants, Dr. Ralph Friedlander, an Associate Member, has also taken part in this work. Before showing you our results, I would stress that the operative procedures in these cases have been done over a period of five years by a changing group of personnel, and while the consultants supervise and sometimes help with the surgery, the actual pre and postoperative care and the operative procedures have been done by a changing group of men, which has included three successive chiefs of surgery and seven junior surgeons who were in process of their training in tuberculosis surgery.

(slide) This is our experience for a five year period from January, 1947, to Jan 30, 1952 a total of one hundred twenty eight resections, with an incidence of bronchopleural fistula increased greatly over the average number in the preceding four years. There are no suitable explanations for this, among which I would cite the fact that we were accepting more and more patients for the major procedures, and also operating on patients whose strepto-

coccal sensitivity had run out. This chart divides the bronchopleural fistulas into right and left. There has been a feeling that fistulas are more likely to occur on the right side, however, our experience has been a little bit to the contrary. The incidence of fistula in the entire series was 10.5 per cent, the incidence in pneumonectomy, 25.5 per cent, is about parallel to that in other types of resections, as is the 6.4 per cent fistula rate in lobectomies. We have had no complication of this type in our small group of segmental resections.

(slide) In thirty four pneumonectomies we had eight fistulas, and, of these, five were complicated by spread or exacerbation of the disease. There were two deaths that occurred in the non-operated patients who had bronchopleural fistulas. In five cases the fistula was still open. In the lobectomy series of seventy four cases there were five fistulas and three associated spread or exacerbations. Two of the patients with fistula, and exacerbation are dead. In two other cases the fistula was closed, and in one case it is still open.

(slide) This shows our total mortality figure for the 125 cases. The mortality in lobectomy was four deaths, 11.7 per cent, in lobectomy three deaths in seventy four cases, 4.1 per cent. Our overall mortality in 125 resections was 5.6 per cent.

We have been impressed with the futility of a direct attack on the fistula in an attempt to close the bronchial stump. Our approach to this complication has been similar to that of Mr. Murphy in his latest group of cases — in early closed drainage with a large tube which is left in situ, either in an intercostal space or through a small rib resection. We use a No. 10 latex tube, and do this drainage as soon as the diagnosis of bronchopleural fistula is made. Our fistulas have occurred largely in the early postoperative period, except in the case which appeared one week after pneumonectomy and was connected with a pocket of pus in the residual pleural space. Following drainage we treat the patient with streptomycin and I.P.V., both parenterally and by injection into the residual space. When the patient is in a good condition, a thoriotomy is planned and performed in stages under

DR LOUIS R. DAVIDSON, New York—I should like to congratulate Dr. Murphy for truthfully telling us what he has told us. In other words, I want to congratulate him for his intellectual honesty. We recently have completed at Seaview Hospital a survey of the results obtained in cases operated upon for pulmonary tuberculosis. It was noted that between February, 1944, and February, 1952, 512 excisions on 487 patients were performed by twenty-one visiting and resident surgeons. As pointed out by Dr. Murphy and his group, we found bronchopleural fistulas to represent the greatest threat to the ultimate welfare of the patients. Eighty-two fistulas have been encountered, this is a 16.2 per cent incidence. However, you will note that in the 512 operations there were 333 pneumonectomies, which is an indication of the type of disease we are meeting with at Seaview Hospital. There were 150 lobectomies and twenty-nine segmental operations.

It was of particular interest to us in collecting these figures to note a definite difference in the incidence of fistulas on each side, as Dr. Mulvihill indicates. Fistulas on the right side are three times more numerous than those on the left. Even the lobectomies revealed the same difference. These fistulas accounted for the greatest number of deaths. In all, there were ninety-seven deaths—the over-all mortality, not from the fistulas, a mortality rate of 19.9 per cent. Of these deaths 31 were due to bronchopleural fistulas with associated empyema. In addition, there appears to be a definite age relationship to the incidence of fistulas, increasing in number with increasing years. We cautiously submit that the answer to this difficulty may be found in a modified treatment of the cartilages, a problem upon which we are engaged at the present time. No definitive statement can be made, but a very solid impression is now held that with this approach to and a consideration of the rigidity of the cartilages, the incidence of bronchopleural fistula has been very definitely decreased.

As to the methods of treating this complication, thirty of these fistulas, or 36.6 per cent, have been closed in the following manner: twelve were closed by bronchoplasty and thoracoplasty, twelve were closed by sodium hydroxide cauterization, five were controlled by completion pneumonectomy, one was controlled by removing the lingula—a completion lobectomy. Of the 31 cases treated with sodium hydroxide, 12 of the fistulas closed, four probably closed, four are still being treated, and the others are definitely unsuccessful.

It is the opinion of our group that a bronchopleural fistula should be treated early.

Sodium hydroxide cauterization should be utilized before extensive empyema has manifested itself. The best results with the use of this agent have been obtained in the smaller fistulas, and especially with a granulation response that occurs after only two or four applications. Where possible, patients with frank empyema should have excisional surgery in the form of a bronchoplasty, a completion lobectomy, or completion pneumonectomy, as the case warrants. This added surgery is unfortunate, but it is perhaps the only way of salvaging patients who, in the great majority of instances, would succumb to the fistula and the resulting empyema. These operations were performed upon 487 patients, there were ninety-seven deaths, 390 patients are living, 208 patients we are now seeing in the outpatient department, forty are being followed by private doctors or other clinics, eighty-three are hospitalized, the great majority of whom have been recently operated upon. You will note that fifty-nine have been lost in the follow-up.

Of these 208 patients, 121 are on full-time activity in various types of employment. A rather unusual observation is interesting—thirty-seven of our patients had forty-one pregnancies. Four of them have had two pregnancies during or since their excisions, three during their pregnancy. Of these thirty-seven cases twenty-four had pneumonectomies, nine had lobectomies, four had segmental resection. Of the thirty-seven cases, thirty-six of these mothers continue in good health and one is dead.

I will close by quoting one sentence from the abstract of Dr. Murphy's paper: "Despite the increased morbidity following development of a fistula, the over-all results of resection have been so favorable as to result in its adoption with increased frequency."

DR WILLIAM M. LEES, Chicago—I think we should at least, while we are talking of the treatment of bronchopleural fistulas, also take cognizance of what causes a bronchopleural fistula. Are we perhaps operating through bronchi that have either been demon-

acted to have a tuberculous bronchitis in the past, or are we operating upon one in whom the tuberculous bronchitis still shows residual evidence of active infection, whether it be mucosal or submucosal? I mention this point because in a series of sixty-five resections, our pathologist has found an overwhelming majority showing tuberculous bronchitis at least in the submucosal layer.

The second point I wish to mention is that I think the old story of bronchial closure will be considered. The use of a noncrushing clamp, if a clamp is used, and the use of the suture material, perhaps 00000 silk on an atraumatic needle, as I have done recently, might be the answer—I do not know. However we have had only five bronchial fistulas, three of which we have been able to take care of with simple collapse measures. It is obvious from the literature that most of the fistulas occur on the right side. Anatomically speaking, on the left side we know the reason why fistulas are comparatively uncommon. The bronchus passes into the aortic window and with one or two sutures we may close the mediastinum completely. On the right side, I think we should be extremely careful to have some form of pleural or bronchial tissue to cover the bronchial stump and perhaps this together with atraumatic sutures may reduce the incidence of bronchial fistulas.

DR. JAMES CARSWELL, IP, McKinney, Texas—Our experience, although much less extensive than that of Dr. Murphy, parallels his results of early fistulas just reported. I would like to bring out the fact that in our hands the treatment of bronchopleural fistula developed following resection for tuberculosis is a much more difficult problem than that faced by a fistula of similar degree in nontuberculous cases, particularly after pneumonectomy. Our experience with secondary closure of a wide open bronchus and plastic obliteration of the remaining space in nontuberculous cases has been very satisfactory and most unsatisfactory in tuberculous cases.

DR. DAVID H. WATERMAN, Knoxville, Tenn.—The disastrous potentialities of bronchopleural fistula were impressed upon us early in 1947 by a case of left upper lobe resection for lung abscess, where the bronchial stump eroded directly into the overlying pleural cavity one month postoperatively. A massive hemoptysis was the first and fatal complication. It occurred to us that the interposition of a more substantial barrier than the layer of pleura we had heretofore used would afford greater protection and promote healing. Since that time we have had only one significant bronchopleural fistula in our series of resections. This series includes only cases where the disease was active at the time of resection and does not include any so-called tuberculoma wedge resections, or cases where a fistula was inadequate follow-up, hence we think the figure is probably significant.

On the grounds that prophylaxis is the best treatment of bronchopleural fistula, I would like to raise the point that has not been laid out of bronchial closure and mention the technique we think may be responsible for the low incidence of fistula. Essentially it is the use of a single continuous suture in the bronchus is divided proximally to the usual interrupted sutures of 00 black silk are placed across the cut end of the bronchus. In the lung left lung. A free graft of pleura is taken from the chest wall and sutured over the cut end of the bronchus using the long bronchial sutures to hold it in place. This method is simplistic itself and has been, we think, most effective.

ond and third ribs, and did an apicolysis, leaving a catheter in the pleural space, with the feeling that the coaptation of the parietal pleura and the raw surface might be helpful in assuring a good sealing of the leak in the bronchus. The patient made an uneventful recovery, and has had no further evidence of fistula.

With this to encourage us, some time later we had a boy who had a very slow air leak for about seven days. We were never able to remove the tube because, although he expanded fairly well, we were afraid to do it. This time, rather than opening the pleural space, we did only an apicolysis and a modified thoracoplasty leaving the catheter in place, with the coaptation of the parietal pleura and the raw surface of the lung. This boy also had an uneventful recovery.

I would like to derive two points from this. One is, that by the conversion of an intra-pleural space into an extrapleural space and the coaptation effect of the parietal pleura on the raw surface of the lung, we may effectively seal off some of these early fistulas. The second is that we were impressed by the absence of shock in these two patients who were opened through the same wound on the fifth and the seventh postoperative days. Wound healing was not delayed, and our results were satisfactory.

DR BECKER (Closing).—I want to thank the discussers for their excellent comments and the moral support that we were given. We are envious of the people who have no fistulas to report and we sympathize greatly with those whose experience is similar to ours. We feel that awareness of a fistula is a very important thing. The diagnoses in some of these cases were not obvious and required painstaking methods to determine that the patient had a fistula. The fistulas in this series were not all early ones. One case was first detected 253 days following the operative procedure. As Dr Murphy said, the earlier the diagnosis and the earlier the active treatment, the better chance of rehabilitating these patients.

I might also comment, since closure was brought up, that we have a series of cases which we compared with our previous series, of patients who were closed by the Overholt technique. We took the twenty previous cases and compared them with twenty we did by the Overholt method, these were pneumonectomy cases. In the previous group we had six fistulas. Using the Overholt technique we had only two fistulas. We feel that this method of closure will prove valuable in treatment of these patients.

In reply to some of the comments that were made, we have also used total thoracoplasties soon after development of the fistula. We believe that the first rib should be removed in these cases, and we also believe that one must have a patient who is fairly well stabilized for him to tolerate the procedure. We have used epidural anesthesia and it has been a great adjunct in this type of therapy. Dr Mulvihill stated that his fistula rate increased in the last sixty-nine cases. I am afraid our experience has been very similar to this. We have taken patients who were more of a risk because of our early results, and we found that our difficulties did increase. As Dr Sanger said, we feel that the prone position is very important, and with that position postoperative spreads have been almost minimal. We have not done concomitant thoracoplasties because we feel that if we can get along without them, we do conserve ventilatory function. We treated one case with sodium hydroxide, as Dr Davidson has recommended. This was a case that had a fistula of fairly long duration and we were surprised when healing occurred, the patient is now discharged.

Concerning bronchial covering, we have used bronchial coverings, but we have found statistically that there was no correlation between our fistula rate and covering or not covering of the bronchus. I would like to show one slide in conclusion, which may illustrate the point that these fistulas are not always apparent.

(Slide.) This is a series of cases that we presented at the Streptomycin Conference in St. Louis in 1951, and it illustrates all the resections that were done in the Veterans Administration Hospital and those done at Oteen. Our fistula rate was higher, 12.3 per cent, but our rate of spreads and our total deaths were much lower. We feel that some of the spreads and the deaths were probably due to undiagnosed fistulas.

PLASTIC SPONGE PROSTHESIS FOLLOWING RESECTION IN PULMONARY TUBERCULOSIS

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IT HAS been known for more than fifty years that under proper conditions and indications a foreign body implanted in the extrapleural space will definitely enhance the efficiency of collapse therapy in the treatment of pulmonary tuberculosis. Various materials have been used for this purpose, including rat, gauze, rubber sheeting, rubber bags, wire mesh, and air. None of these proved particularly satisfactory until Baer in 1913, reported the use of paraffin. This material for years enjoyed the greatest popularity and was generally accepted as the material of choice to be used when plombage was indicated. This popularity continued until Wilson and Baker,¹ in 1946, reported their experience with the implantation of methacrylate molds in the extrapleural cavities of forty dogs. Wilson² later reported the results of the implantation of these molds in the extrapleural cavities of fifteen patients with pulmonary tuberculosis. Methyl methacrylate (Lucite) enjoys certain properties which many workers felt gave it a distinct advantage over paraffin. It is lighter, produced less tissue reaction and was much easier to handle. It also possessed certain disadvantages which do not come within the scope of this paper.

As resection surgery became better developed and it was possible to remove the entire lung with a fair degree of safety, certain new problems presented themselves. It was soon realized that when an entire lung was removed in a case such as bronchogenic carcinoma a large dead space was left resulting in a shifting of the mediastinum to the operative side and in overinflation of the remaining lung. The overexpanded lung following pneumonectomy has been shown by Cournand and his co-workers^{3,4} to reduce the respiratory function and reserve in both adults and children. In some cases cough, pain and dyspnea occurred following large resections. Evacuation of the dead space has been accomplished by phrenic section and pleurotomy. One other method was to allow the cavity to fill with a serous exudate which in time became organized.

It does not seem strange, then, that with the experience gained in extra pleural implantations of foreign bodies some type of prosthesis might be indicated in the pleural space after pneumonectomy. Johnson and his co-workers,⁵ in 1947, reported the use of a large, hollow metal prosthesis of zirconium and also the implantation of multiple small, hollow plastic balls of Lucite. Small, Clagett, and Grindlay⁶ reported on the use of a gelfoam prosthesis in 1947. This proved unsatisfactory because as time passed the gelfoam was completely absorbed. They also tried a polyethylene prosthesis which was more or less flexible and which was filled with glass wool. Each of these materials had some drawback. In 1950, Grindlay, Clagett, and Rydell⁷ reported on the use of a plastic prosthesis which was made to a size roughly that of the pleural space of the removed lung. Grindlay and Waugh,⁸ in 1951, reported on their experimental and clinical experience utilizing the Ivalon sponge as a frame work for living tissue. These authors used the sponge in various ways unrelated to thoracic surgery, and, in addition, implanted sponges in the pleural cavities of eleven dogs following pneumonectomy. None of the dogs showed any systemic reaction. Examination of these sponges two to eighteen months after implantation revealed that there was firm fixation to the chest wall, while on the diaphragmatic surface the fibrous capsule of the sponge was covered with a smooth and glistening layer of pleura. The mediastinal surface was loosely adherent to the structures of the mediastinum. Microscopic examination of removed sponges revealed no inflammatory reaction, the sponge spaces were occupied by cellular tissue and bundles of collagen fiber around large spaces, the walls of which were lined with what resembled a single layer of vascular epithelium. Levine and associates,⁹ in 1951, reported the implantation of several substances in the extrapleural space of dogs. These authors used vinyl copolymer, polystyrene, cellulose, and neoprene. Each was discarded because of some disadvantage, ranging from lack of uniform physical properties to toxic effects. Levine and co-workers, like Grindlay and Waugh, found the Ivalon sponge the most satisfactory in the experimental group. Hurst and collaborators,¹⁰ in 1951, reported the clinical use of Ivalon in extra pleural pneumonolysis in fifteen instances. Two patients had insertion of the sponge after resection, but no mention was made as to whether it was placed intra- or extrapleurally. Charbon and Adams¹¹ used the Ivalon sponge in dogs to prevent overdistention of the remaining lung following resection. They concluded that the Ivalon sponge was unsatisfactory as a prosthesis since it became hard as a stone, and its adherency to the pericardium might interfere with cardiac action.

There are certain requirements which have to be met before any foreign body is placed in the tissues or utilized as a prosthesis. The material should be nonirritating, chemically inactive, stable, and produce minimum foreign body reaction. It should maintain its size and shape, be light in weight, easily removed, and, very important, it should be elastic.

Following the favorable report on Ivalon by Grindlay and Waugh, which suggested that it approached the above criteria better than any material pre-

ously used, we undertook to repeat their experiments. Furthermore, we compared the reaction produced by Ivalon with other agents commonly buried in body tissues.

Our findings corroborated those of the Mayo group. When the sponge was placed intrapleurally it became adherent to the visceral and parietal pleural surfaces in the form of a thin layer of mesothelial tissue. On palpation the sponge had an India-rubber consistency (Fig. 1).



Fig. 1—Sponge intrapleurally—sixteen weeks postoperatively. Visceral and parietal pleurae adherent to the sponge by mesothelial tissue. The sponge is in the center with partially expanded lung above and the chest wall below.

On cut section an areolarlike tissue was noted between the sponge and surrounding host tissues. This appeared less marked on the lung side, and a minimal reaction was observed about the visceral pleura. This paucity of reaction permitted a rapid development of a cleavage plane and the visceral pleura was stripped readily from the sponge without injury to the pulmonary tissue. In the cross section (Fig. 2) it can be seen that the India rubber consistency is due to an ingrowth of connective tissue extending into the spaces of the sponge for varying distances up to 1 cm. This invasion of the sponge by connective tissue gave the characteristic appearance of a central spongy liver and a denser peripheral one. Microscopic sections (Fig. 3) showed a very mild or no reaction in either of the pleurae or the lung. However, a small amount of fibrous tissue could be seen filling the peripheral spaces of the sponge. The same general results were obtained when a sponge was implanted intrapleurally. Unfortunately, difficulty was encountered in developing large peripheral spaces in dogs. Because of this it was necessary to compress the sponge in order to insert it into a relatively small bed. Sixteen weeks later the sponge was removed; it appeared flattened and somewhat dense, but still had an India rubber consistency (Fig. 4).

On cut section blood was readily expressed from the tiny capillaries when the sponge was gently squeezed between the fingers. The areolar tissue between the sponge and surrounding tissue was so pliable that the intercostal nerve adjacent to the sponge moved freely when traction was applied. Peeling the sponge from the adherent structures was relatively simple (Fig 5).

These observations would indicate that the Ivalon sponge produced little or no reaction in the host tissues and that its density was modified by the de

Fig 2

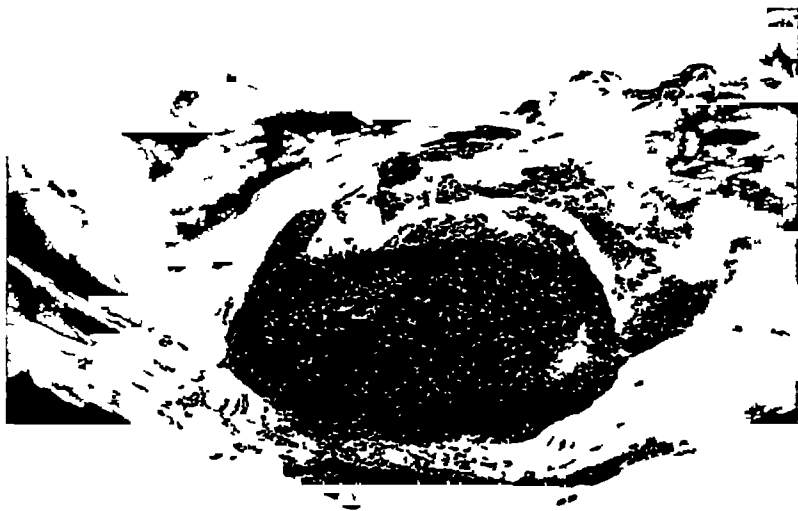


Fig 3

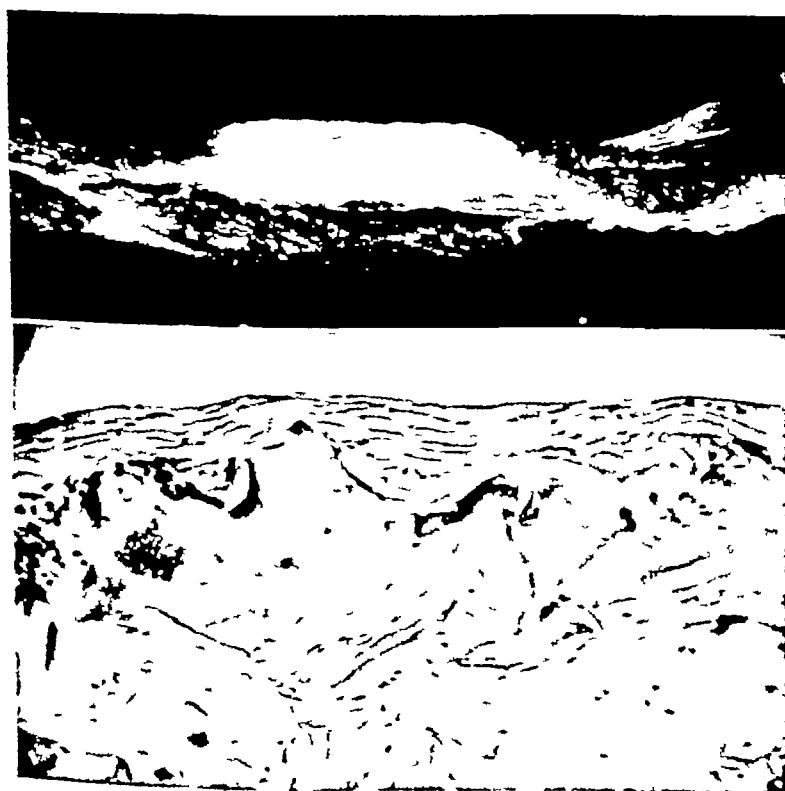
Fig 2.—Sponge intrapleurally—cut section—sixteen weeks postoperatively. The sponge is in the center with lung above and chest wall below. Note central spongy layer and denser outer layer in the sponge. Denser area is produced by an ingrowth of fibrous tissue. No fibrotic reaction of tissues is present about the sponge.

Fig 3.—Intrapleural sponge—microscopic section sixteen weeks postoperatively. Lung shows no evidence of inflammation or fibrosis.



FIG. 4—Sponge extrapleurally—sixteen weeks postoperatively. No adhesions to lung. Lung has become vascularized. Irregularity in illustration demonstrates plastic sponge conforming to the spaces of sponge surface.

Fig 5



gree of fibrous tissue growth into the sponge, the extent the sponge was compressed by its bed, and compression of the sponge while hot in the process of sterilization. Perhaps the described stonelike character of the sponge occurring in Charbon and Adams' experiments could be explained by one of these factors.

Interference with heart action with this prosthesis would be quite unlikely since the fibrous reaction is almost completely limited to the sponge, no intrapericardial adhesions are formed, and the area of pericardium involved in the tissue reaction is considerably less than one half of its entire surface.

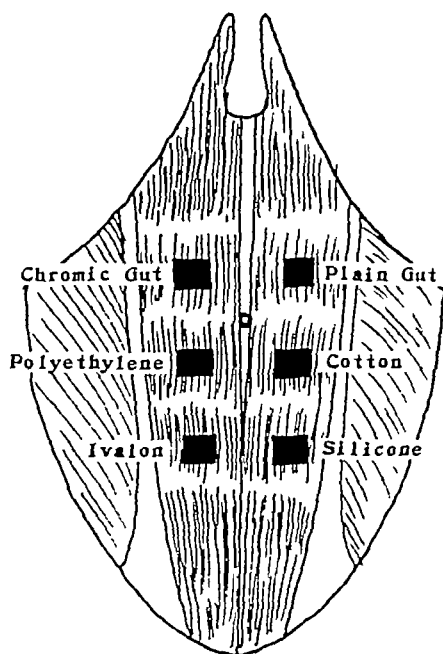


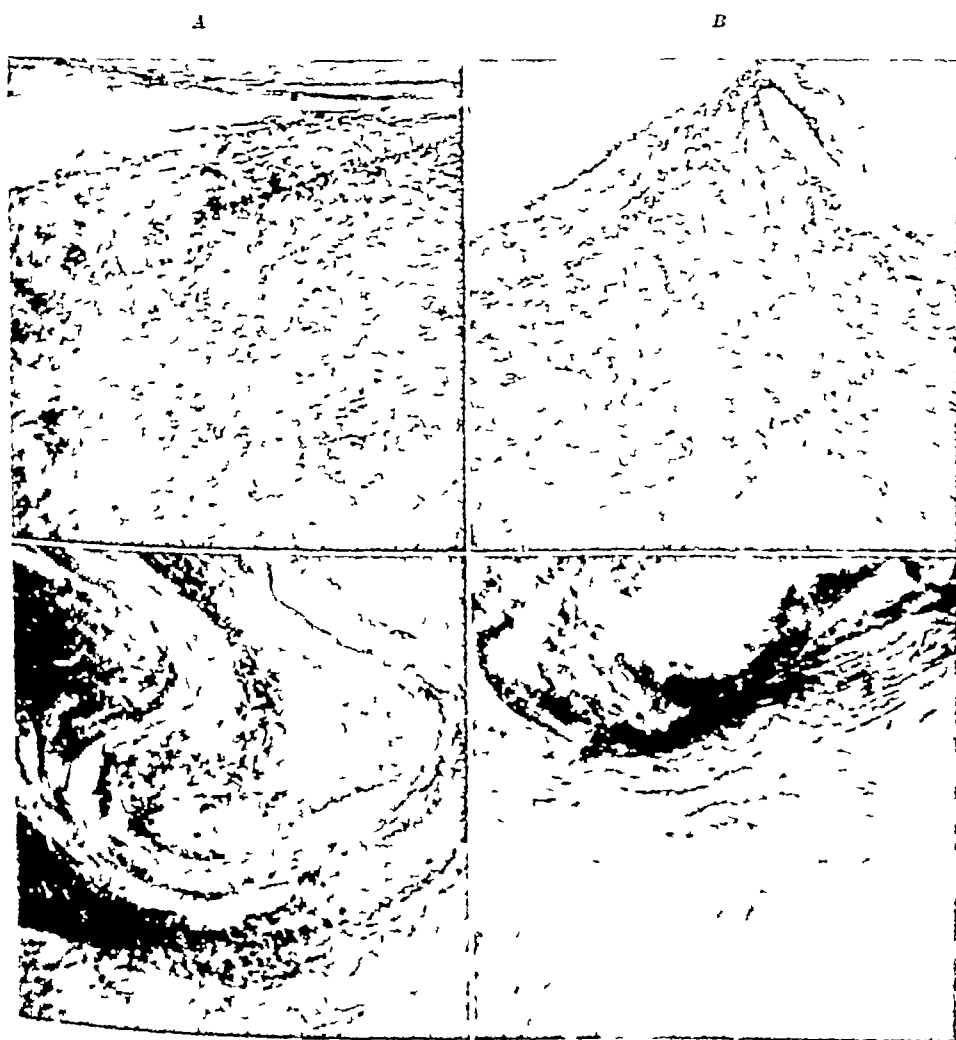
Fig 7—Diagram showing where implanted materials were inserted into anterior abdominal wall of a dog

The comparative study of Ivalon sponge was made with plain and chromic catgut, polyethylene, cotton, and silicone. These substances, cut in small sheets 2 by 2 cm, were inserted through individual incisions in six different areas of both rectus sheaths of dogs (Fig 7). The dogs were sacrificed at different times. Both recti and their sheaths were excised in their entirety, and blocks made from tissue contiguous to the implanted experimental materials.

As anticipated, the plain and chromic catgut produced a violent inflammatory reaction both grossly and microscopically in the first and second weeks (Fig 8). By the fourth week the inflammatory exudate was replaced by fibroblasts which were beginning to lay down collagen. By the tenth week there was definite scar tissue. The sheets of gut could be seen in all the sec-

was, but by the tenth week there was definite fragmentation. At forty-six weeks (ten and one-half months) only scar tissue was found, and all catgut had been absorbed.

Cotton, when employed in sheets, had a tendency to migrate from its bed either subcutaneously or to the peritoneum. When subcutaneous migration occurred, additional inflammatory reaction was noted. In general there was



D

much less inflammatory reaction than with the catgut, and fibroblasts were observed about the cotton fibers at the end of one week (Fig 9) At six and ten weeks (Figs 12 and 13) the presence of monocytes, fibroblasts, collagen, and capillaries about the cotton fibers suggested a moderate to heavy foreign body reaction

Polyethylene manifested minimal inflammatory reaction, although in the acute phase considerable edema was seen This edema disappeared, and from

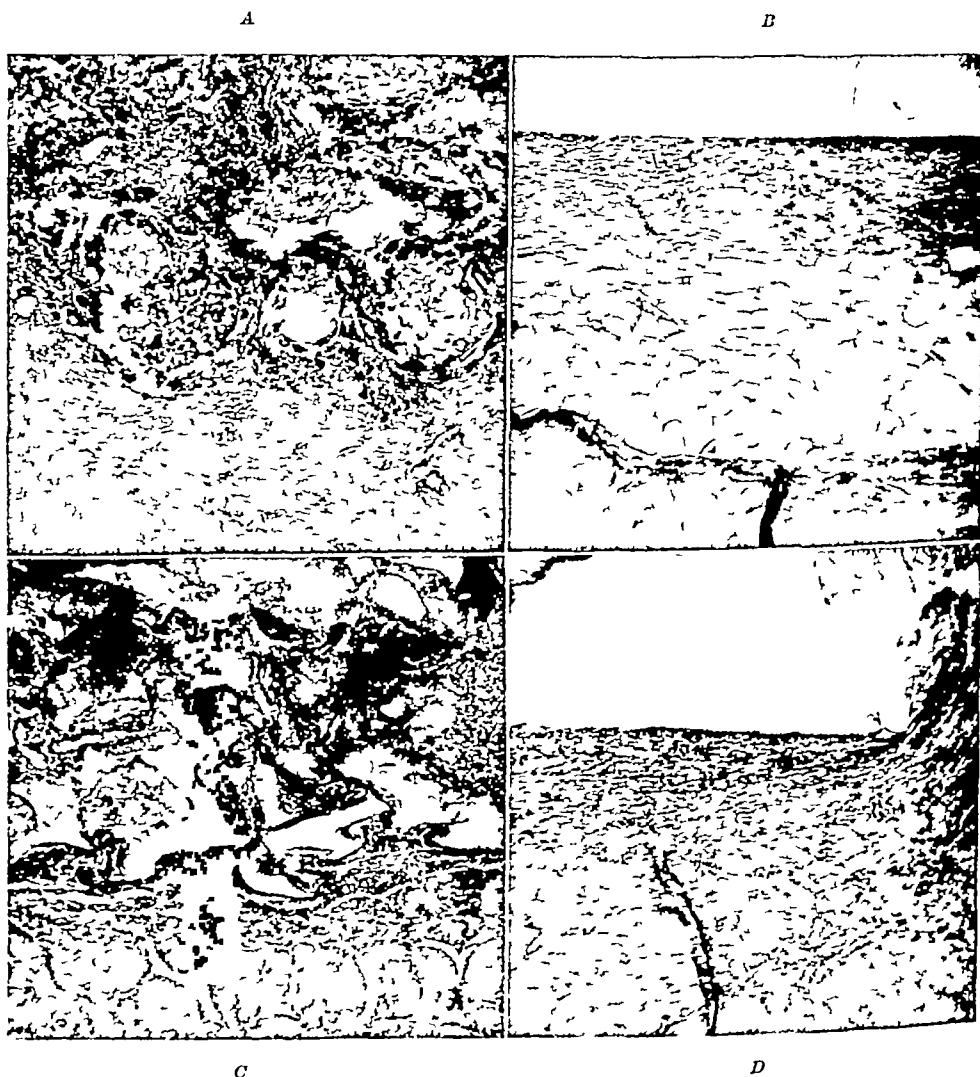


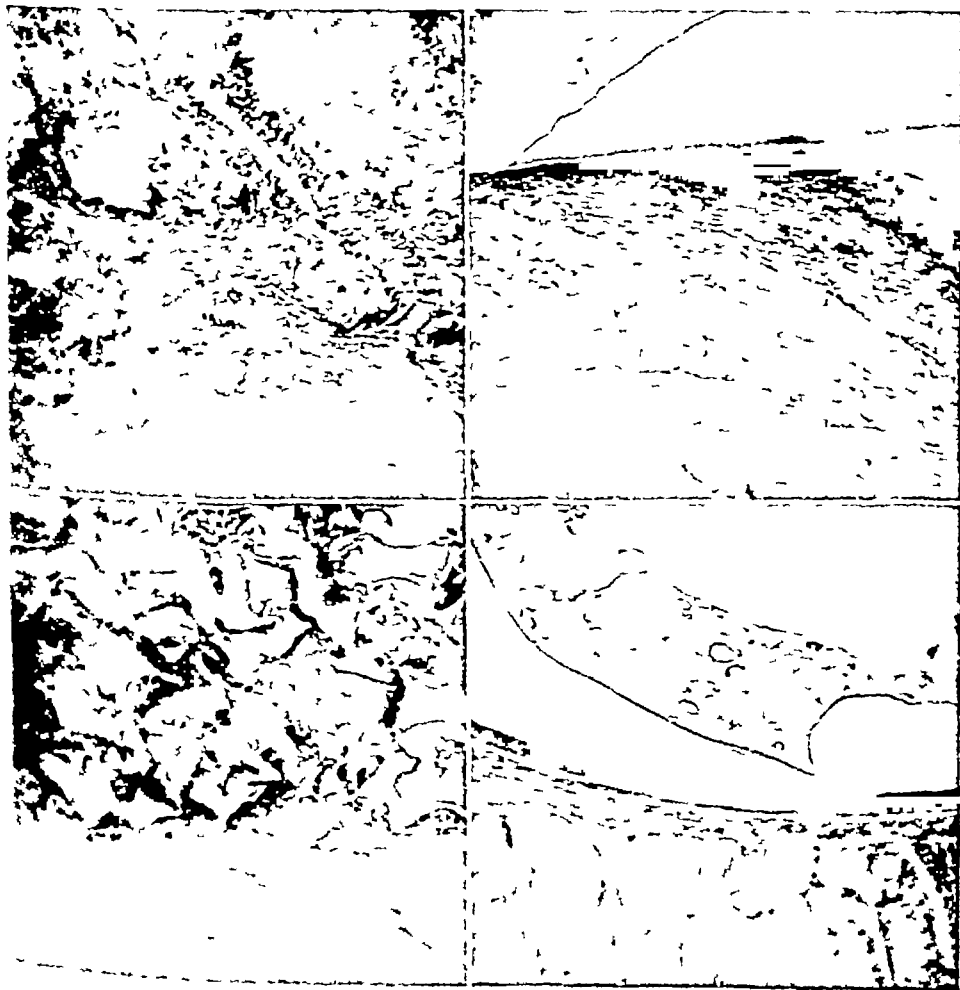
Fig 9—Cotton polyethylene Ivalon and silicone implanted in the rectus sheath—one week postoperatively A Cotton—A moderate amount of inflammation with fibroblasts developing around cotton fibers B Polyethylene—The polyethylene was in contact with tissue at the upper end of the illustration but fell out at time of section Note moderate amount of fibroblastic activity Inflammatory reaction was minimal C Ivalon—Some inflammatory cells present about the muscle fibers Note growth of capillaries and fibroblasts into pockets of the sponge D Silicone—There is a thin layer of inflammatory reaction and fibroblasts where silicone had been in contact with muscle at the upper end

the second to the tenth week (Figs 10, 11, 12, and 13) a fairly thin layer of fibroblasts matured into a thin layer of dense fibrous tissue containing considerable collagen.

Ivalon in its first week exhibited only inflammatory cells between the muscle and the sponge, while fibroblasts and capillaries were growing into the pockets of the sponge. By the second week there was no evidence of inflammation and the vascularized fibroblastic activity proceeded into the meshes of

A

B



D

the sponge. This ingrowth of fibroblasts continued during the ensuing weeks. At six weeks areolar tissue containing blood vessels was noted at the junction of the Ivalon sponge and the host tissues, and by the tenth week this was more pronounced.

The least reaction was visualized with silicone. Practically no inflammatory response was observed. A very narrow rim of fibrous tissue later developed about the silicone.

At forty-six weeks the gross appearance of polyethylene, cotton, Ivalon, and silicone-implanted areas showed no reaction. All implanted substances were surrounded by loose areolar tissue.

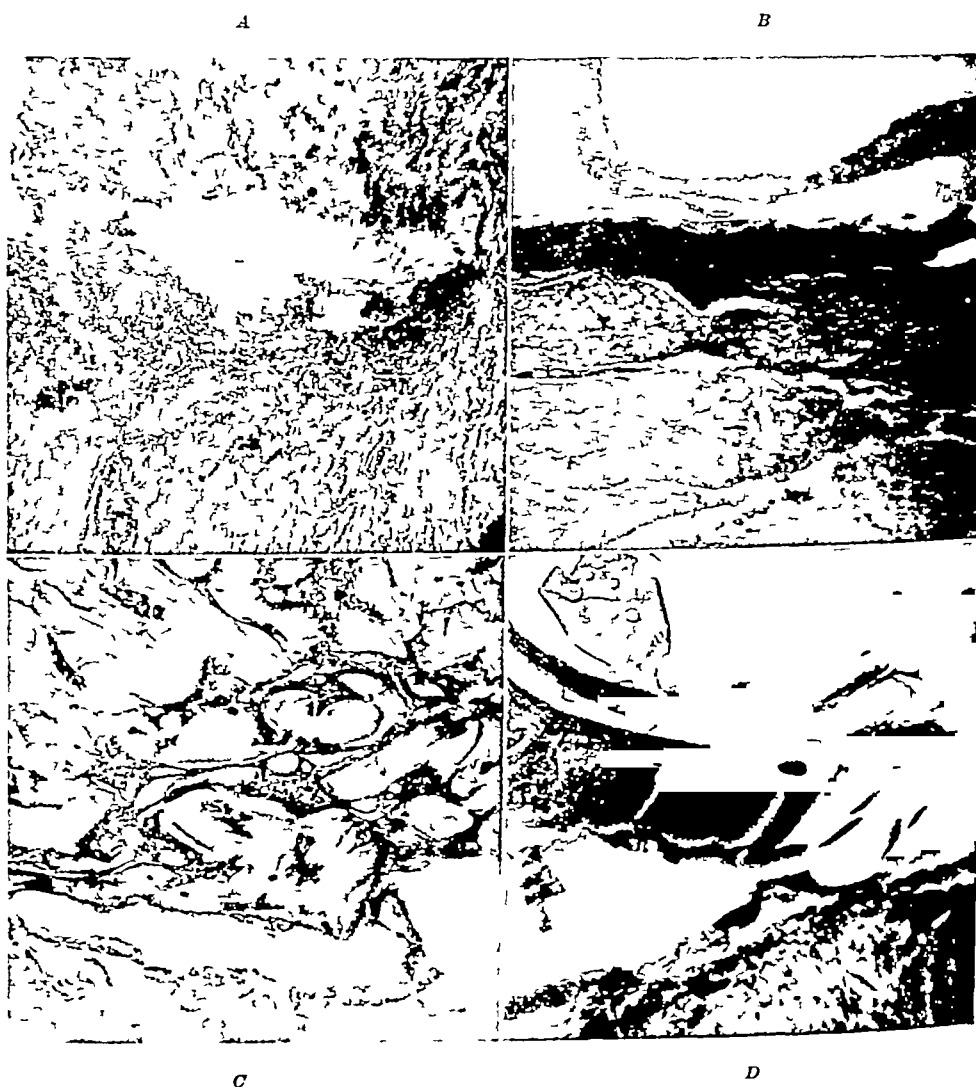
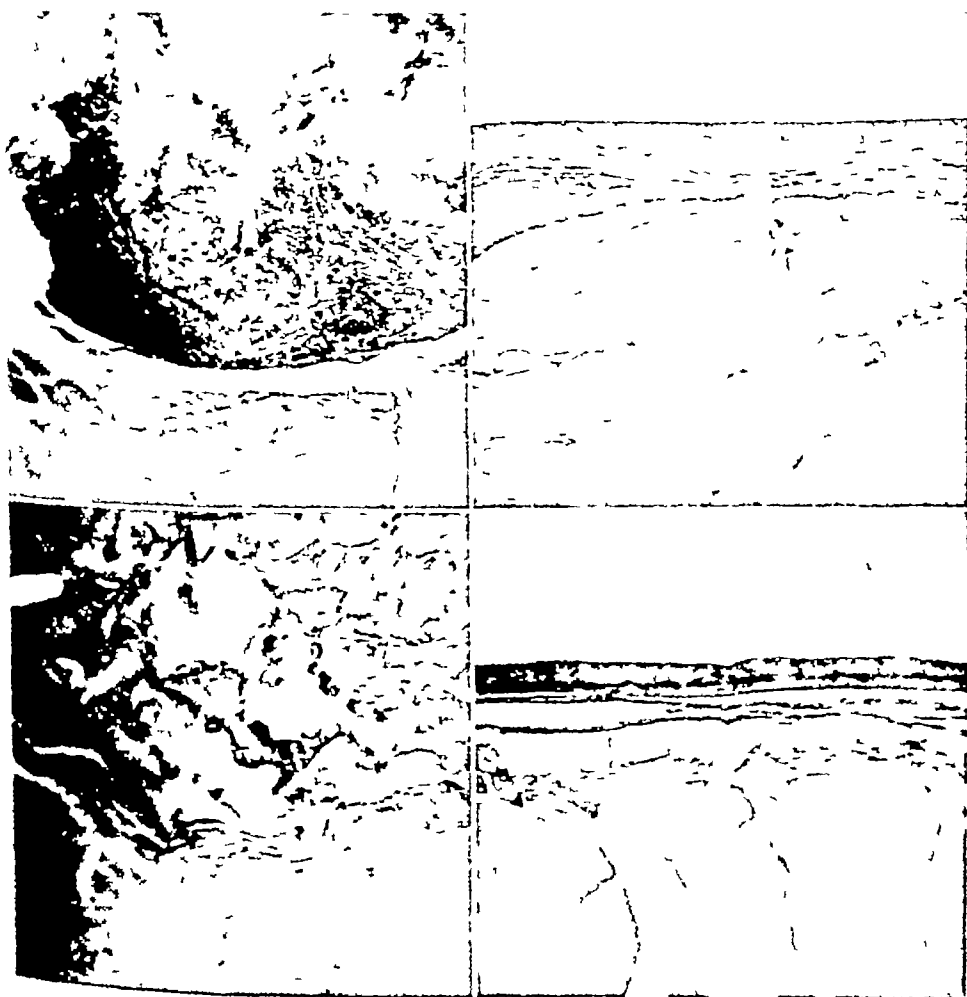


FIG. 11.—Cotton, polyethylene, Ivalon, and silicone implanted in rectus sheath—four weeks postoperatively. *A* Cotton—Reaction is similar to the one which was seen at two weeks. *B* Polyethylene—Presents slightly more fibroblastic activity than at two weeks. *C* Ivalon—No inflammatory reaction. Vascularized fibrous tissue extends at least 5 mm. into the sponge. *D* Silicone—Rim of fibrous tissue seen extending into the muscle.

The microscopic sections (Fig 14) at ten and one half months were difficult to cut since the tissue reaction was gone and the implanted material slumped about in the areolar tissue. Ivalon, polyethylene, cotton, and silicone demonstrated practically no reaction in the adjoining muscle tissue. Between the muscle and polyethylene, silicone, and Ivalon, areolar tissue was seen. No areolar tissue was observed with cotton. In the meshes of Ivalon dense fibrous tissue, collagen, and blood vessels were noted. Silicone showed the least reaction with a very fine layer of fibrous tissue.

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B



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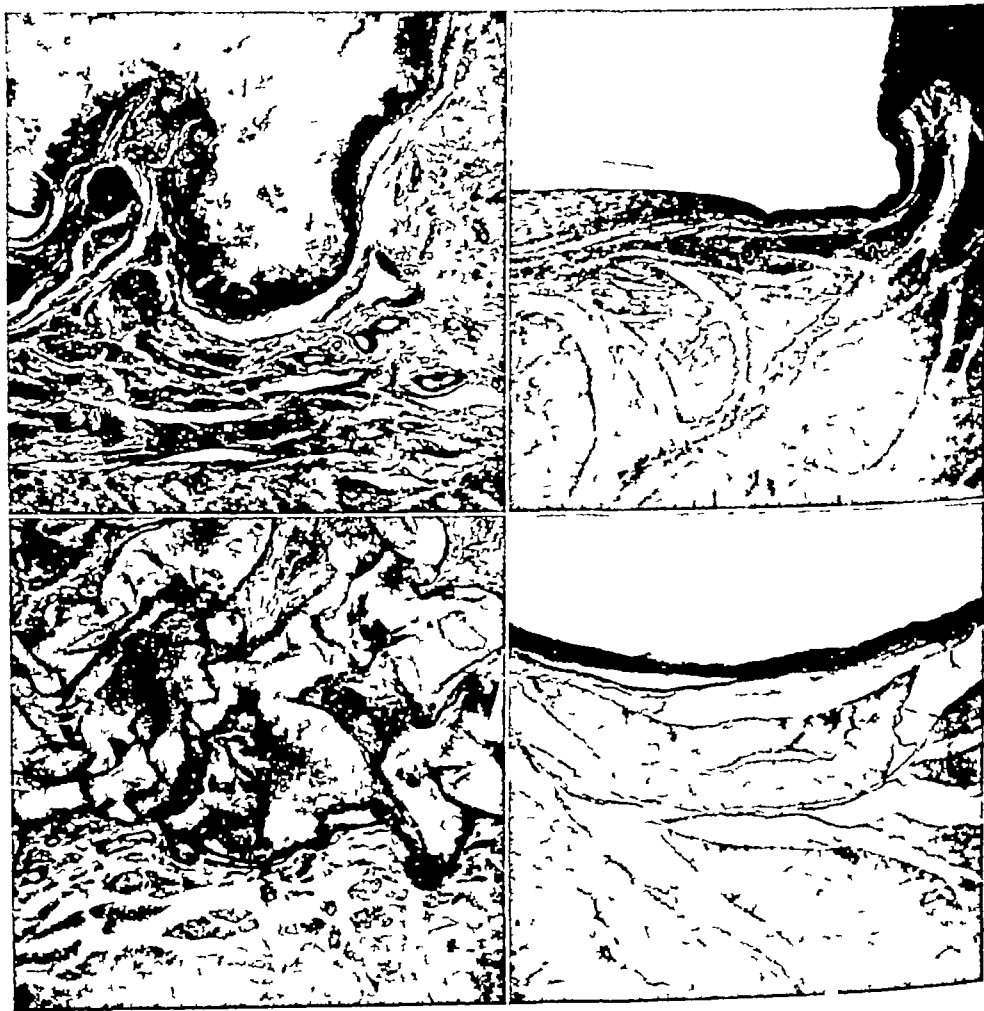
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The polyvinyl sponge, if pure, is white in color, odorless and tasteless, and the cut surface presents the appearance of a piece of white bread. It is quite tough, and when dry becomes hard. When moistened it becomes very soft, flexible, and elastic. It is very easy to cut with scissors when soft. It is light in weight when dry, with a density of 0.05. The sponge absorbs water freely, increases in size when saturated, and on drying there is slight shrinkage.

Grindlay and Waugh found that temperatures of 120°C had no effect on the sponge, but after being exposed to 160°C for an hour it began to turn brown. Slight shrinkage occurred after boiling for the first time, but re-

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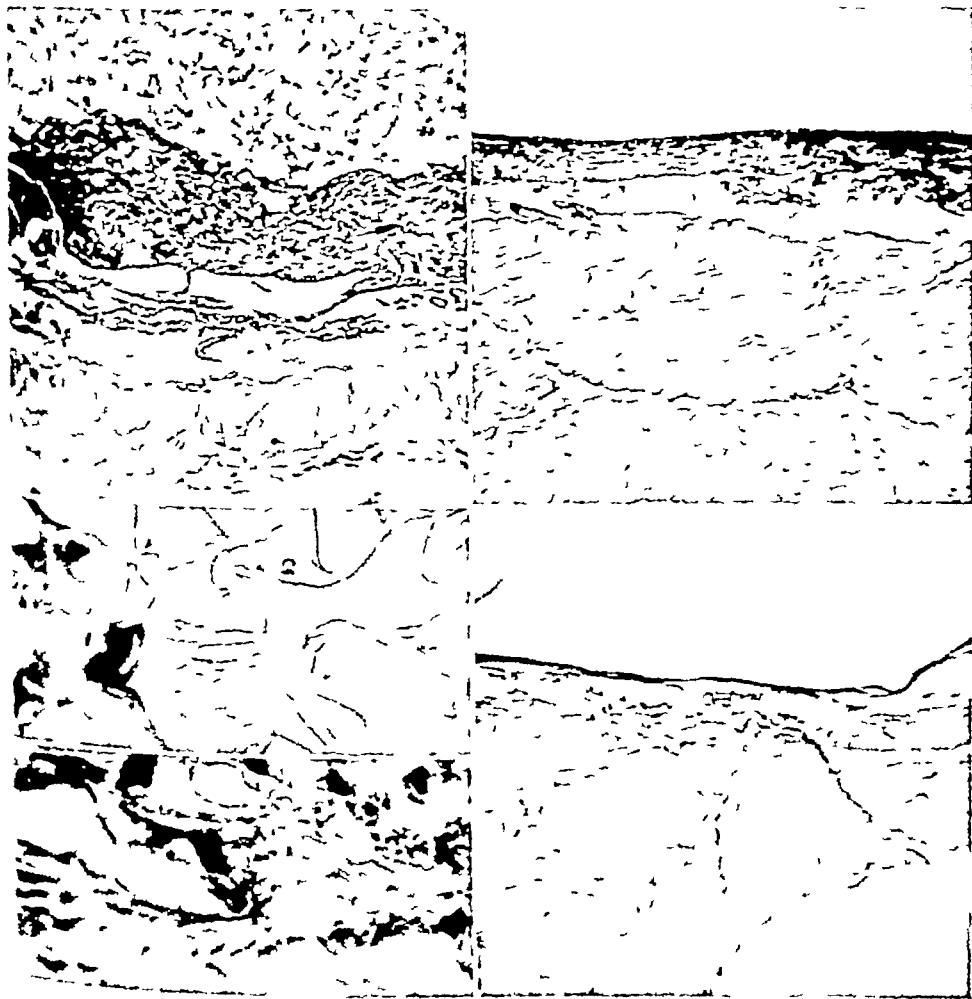
Fig. 13—Cotton, polyethylene Ivalon, and silicone—implanted in the rectus sheath—ten weeks postoperatively. Changes about all four implanted agents are the same as at six weeks.

peated boilings did not affect its size. They also found that if the sponge was compressed or shaped when subjected to a temperature over 95°C , the shape would be maintained when cooled.

The sponges are available in two sizes: 17.5 by 9.5 by 6.5 cm and 33 by 19 by 14 cm (Fig 15). We prefer having both sizes available for use. Each sponge comes from the manufacturer packed in an airtight plastic bag which

A

B



C

D

keeps it moist and soft. Upon removal from the bag the sponge should be thoroughly washed to remove the hygroscopic substance added by the manufacturer. This is easily done by squeezing the sponge several times under running water. Sterilization is simple. The sponge is placed in a clean vessel with a snug-fitting lid. It should have ample space to fit into the pan without deforming. It is boiled for thirty minutes. It may be cooled slowly, or quickly by adding cold saline solution.



Fig. 15—Photograph showing the two sponge sizes: small—17.5 by 9.5 by 6.5 cm. and large—33 by 19 by 14 cm.

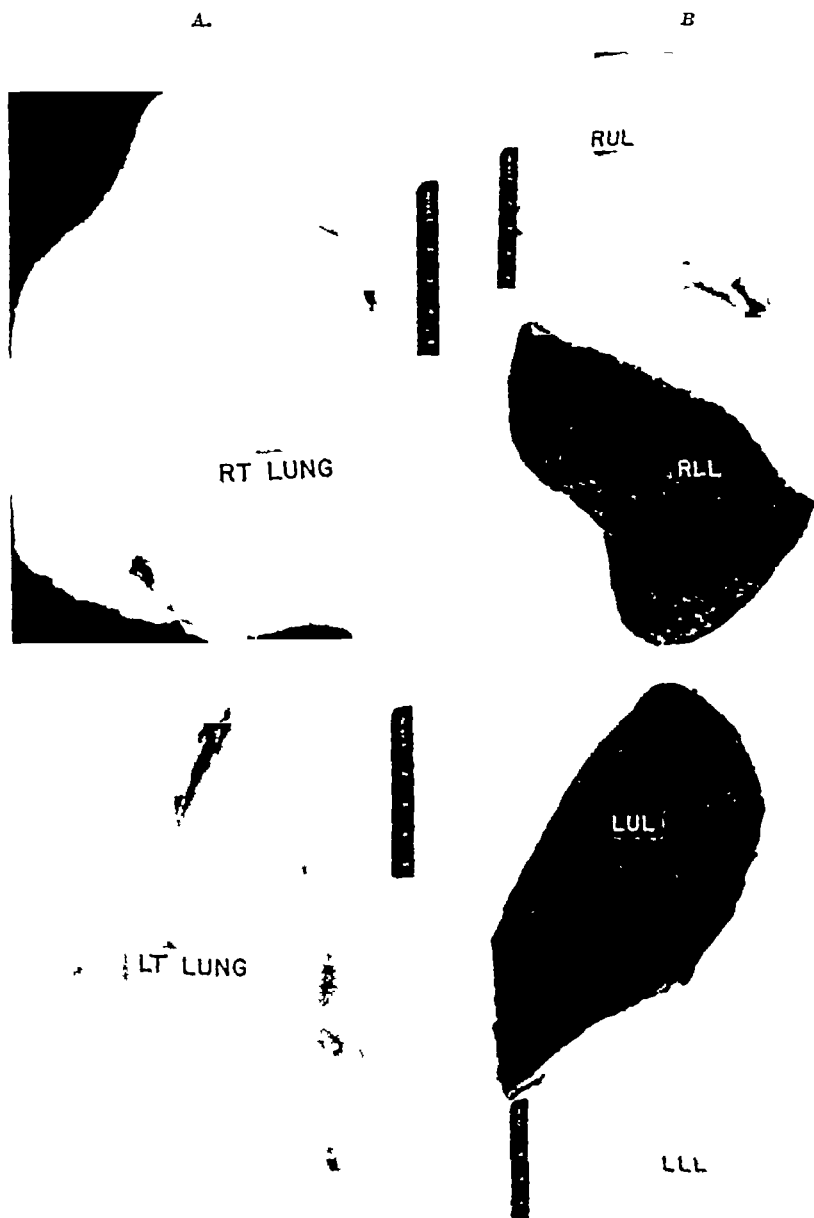
At the time of surgery it has been our practice to remove the sponge from the sterilizing vessel, wring it out thoroughly after soaking in normal saline solution, and then to cut it to the shape desired using heavy suture scissors. Recently the manufacturers have molded the sponges into the shapes of a right and a left lung and separate lobes (Fig. 16). These are more artistic and need less sculpturing before placing into the intra- or extrapleural space. There is also less waste of time and material involved. The expense is the only deterrent to their use. Time will tell whether the added expense will be worth while.

Since April, 1951, we have utilized the Ivalon sponge as a prosthesis following lobectomy, pneumonectomy, and segmental resection in fifty cases (Table I). It was used at the time of operation in thirty-three instances, and following operation seventeen times. Twelve sponges were placed intra

TABLE I

OPERATION	CASES	INSERTION OF SPONGE			
		AT TIME OF OPERATION		LATER	
		INTRAPLEURAL	EXTRAPLEURAL	INTRAPLEURAL	EXTRAPLEURAL
Pneumonectomy	8	1	2	4	1
Lobectomy	34	4	19	2	9
Segmental	5	1	6	0	1

pleurally and thirty-eight extrapleurally (Figs 17, 18, and 19) We have not felt it necessary to use the sponge following lobectomy where the involved lobe is small, crenated, and atelectatic, and when the contiguous lobes have already overexpanded to occupy 85 to 90 per cent of the pleural space



In most instances the filling has been extrapleural when done at the time of the resection. We have felt rather timid about placing a foreign body in the pleural space where we knew tuberculous lesions had been molested. In

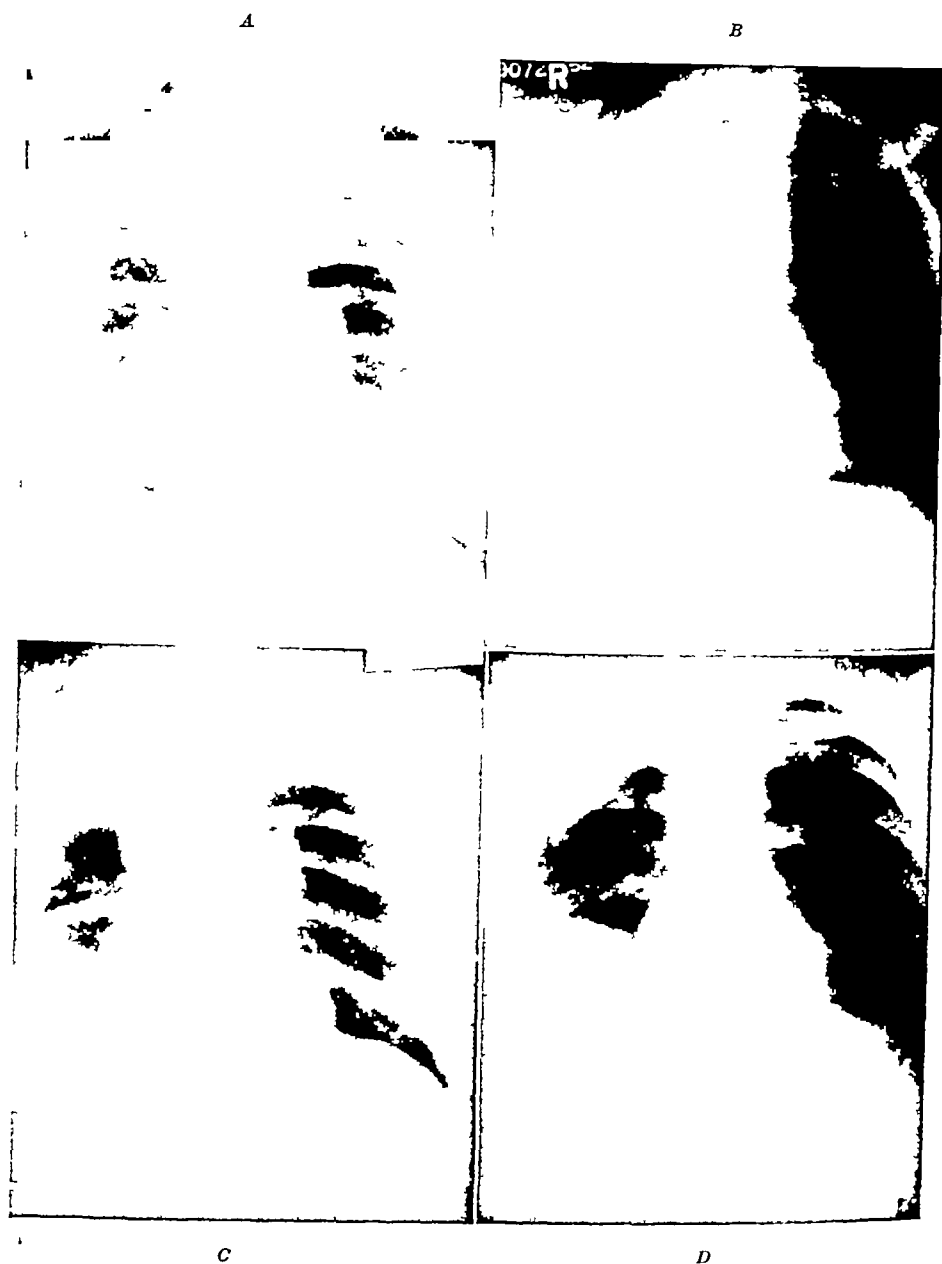


Fig 17—A Preoperative film on patient with bilateral apical tuberculosis. B Film taken four days following right upper lobectomy. The apparent large size of the sponge is due to fluid. C Film taken two months later. Fluid has absorbed and lower lung field has cleared. D Film taken four months after lobectomy. The size of the sponge about the same as at two months. Diaphragm high phrenic not disturbed. Diaphragm usually returns to normal position.

these cases, following the removal of an upper lobe the parietal pleura is dissected free from the chest wall and mediastinum down to the remaining lobe. In an extrapleural dissection of the cupula was necessary to mobilize the resected lobe, the defect can be repaired easily after dissection and mobilization

1

B



C

FIG. 1. Four radiographs of patient with bilateral hilar lymphadenopathy and extrapleural inversion of the upper lobes. (A) Preoperative, (B) postoperative, (C) postoperative, (D) postoperative. The sponge is cut to size, care being taken to make it slightly larger than the lung. This seems important because the sponge not only expands when it absorbs blood and serum, but tension and traction

If the sponge was not inserted at the time of resection, it is a rather simple matter at a second operation to peel the parietal pleura away from the chest wall and place the pack extrapleurally. This was done in eleven cases.

The sponge has been placed intrapleurally at the time of operation in only six cases. This has been done without mishap, but our experience is not

A

B



C

D

Fig 19—1 Patient ten days following right upper lobectomy. B Same patient twenty-four hours after insertion of sponge at right apex. C Appearance six months later. All reaction has subsided. D Appearance one year later. No change noted since previous film taken at six month.

great enough to recommend it. One might postulate that following pneumonectomy particularly it might increase the hazard of bronchial fistula which occurs too often in tuberculous cases. In pneumonectomy, where the phrenic nerve is cut at the time of operation, one cannot be sure how high the hemidiaphragm will rise and what size the sponge should be. If it were too large, pressure, friction, and complications might ensue.

In patients in poor condition, or if an extensive extrapleural dissection had been necessary for lung mobilization, and following pneumonectomy, we prefer to insert the sponge intrapleurally after waiting seven to ten days. A portion of the wound is reopened, and the pleural space is entered either through the periosteal bed of the resected rib or by resecting a portion of the fourth rib. The dead space is irrigated thoroughly with saline to remove the old clots, and the size of the cavity is determined. A large opening in the chest wall is not required since the compressible sponge can be forced through a small opening without difficulty.

As we have gained experience it appears that it makes little difference whether the sponge is placed in either the intra- or the extrapleural space at the time of the second operation. We now feel that the intrapleural technique would appear preferable since the time consumed is much less, the procedure very simple, and the results excellent.

When the sponge is inserted at the time of resection, whether intra- or extrapleurally, there is always some fluid accumulation. This is apparently not due to the foreign body reaction but appears to be in direct proportion to the extent of dissection required by the resection. The sanguinous exudate is rarely completely evacuated following lobectomy even with continuous suction. This has been found true in every instance where the pleura was opened several days following a resection. If the fluid accumulation becomes excessive following the insertion of the sponge, a thoracentesis can be done. Five patients were aspirated, but in only two of them was it repeated. Each of these necessitated three aspirations. In the remaining three cases which were aspirated once, the procedure proved unnecessary, for in later cases the fluid was found to absorb gradually as it does following Lucite or paraffin drainage.

It has been interesting to watch the roentgenograms of these patients during the ensuing months. The sponges enlarge at first but as the exudate and infection subside they appear to shrink to a size even smaller than at the time of insertion (Fig. 17). This change does not seem to be great enough to detract appreciably from the efficiency of the plompage. It certainly is more satisfactory and less deterring than a thoracoplasty.

In this series of fifty cases there were no complications in forty-six. The sponges have been in place for one year, twenty-six for six to twelve months, and twelve for less than six months. It was necessary to remove the sponges in three instances. One patient, a diabetic, had a right upper lobectomy and extrapleural filling at the time of the resection. He developed a pneumonia due to the hemolytic *Staphylococcus* and an extrapleural abscess. The sponge was removed twelve days later, drainage established,

and later a thoracoplasty performed to obliterate the dead space. The second patient had an unexplained fever following a resection and simultaneous sponge placement. Three aspirations revealed sterile fluid. The fever subsided and the patient returned to the sanatorium with orders to continue his streptomycin and PAS therapy. This was not done, and four weeks later pus began draining from the wound. A mixed infection empyema was drained, antibiotics administered, and later the space was obliterated by a thoracoplasty. These two patients must be considered as failures. The infection in the diabetic patient was the chief offender. In the second instance, probably infection, but certainly the error in not continuing the antibiotic therapy may have contributed to the complication.

There were two postoperative deaths. One patient died eighteen hours after a segmental resection of the right upper lobe. His condition was excellent until 3 A. M. when his roommate reported his respirations were noisy and labored. He died rather promptly. At postmortem examination a tension pneumothorax was found on the opposite side. The second patient died six days after a lobectomy from a transfusion reaction. Neither of these deaths can be attributed to the sponge fill.

SUMMARY

Fifty cases of lobectomy, pneumonectomy, and segmental resection have been reported in which the polyvinyl sponge has been used as an intra- and extrapleural plombage.

By utilizing this type of prosthesis, the dead space created following resection is filled and accompanied by a minimal amount of reaction. This prevents the overexpansion of the remaining lobe or lobes on the operated side. Thoracoplasty, heretofore necessary to accomplish this, is eliminated, and the contour and functional integrity of the thoracic cage are preserved.

It is possible to complete the procedure at one sitting. If this is not advisable and a second operation is necessary to insert the sponge, it is simpler and less shocking than a thoracoplasty. We feel that in those cases where further resection is necessary on the opposite side, the patient will be a better operative risk with an intact thorax and without overdistention.

We are fully aware that as more is learned about the new drugs and their therapeutic use in tuberculosis, the amount of lung tissue which has to be sacrificed at operation will be greatly decreased. During the past year we have performed two hundred resections for tuberculosis, and 48 per cent have been of the segmental type. Many of these have consisted of the removal of such small segments that any type of fill is unnecessary. If it had not been for these changing trends our series would have been larger. Until one year ago we have found that in over four hundred resections approximately 95 per cent were lobectomy or pneumonectomy.

Any foreign material placed in the body is not ideal and will always have disadvantages. If these are minimal when compared to the beneficial effects, as demonstrated in this small group of cases, it would appear that we are justified in recommending the procedure.

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DISCUSSION

DR OTTO C BRANTIGAN, Baltimore Md.—I would like to congratulate Dr Gale on his beautifully presented work and to add my brief experience with pulmonary prostheses. Following the report by Julian Johnson on prosthesis with Lucite spheres, we later at one in 1947 and have used the method on 33 patients. The method has been used only in the patient who had pneumonectomy for tuberculosis. The patient is allowed to recover from the pneumonectomy and at a later date the Lucite sphere prosthesis is placed into the intrapleural space. The prosthesis is applied under local anesthesia when the chest wall has reached the desired position. Unfortunately three patients of the group developed infection in the pleural space about the sphere—one pure tuberculous infection and two pyogenic infections. In seeking a cause for the infection it was found in each instance there had been gross contamination of the pleural cavity at the time of the pneumonectomy, and the contaminated pleural space had been treated by therapeutic measures sufficiently to avoid an initial empyema. There were other patients who had gross contamination at the time of pneumonectomy who have had successful prosthesis without infection. The patient who had the pure tuberculous infection and the prosthesis actually had a pleurectomy and pneumonectomy for tuberculous empyema. The prosthesis should not be used in the pleural space when gross contamination has existed at the time of the pneumonectomy. The procedure has not been used for the patient who has had lobectomy. There have been no other complications. The first patient with the prosthesis almost five years and he is quite well and free of trouble. There is no way to know how long he will be at the end of twenty or more years. I have found that there is a tendency for Lucite spheres to wear through where they come in contact with each other. Wilson had the foresight to insist on having the sphere on the inside.

Inasmuch as I had not contemplated discussing this paper, I do not have at hand my exact statistics. However, we have used sponges in about 25 patients and it is our experience in these cases that I wish to give you. In one patient who had a right lower lobectomy, the drainage tube did not function properly, and a few days later the tube was removed and decortication performed. The sponge was resterilized and replaced. Subsequently the patient developed an empyema and later died of progression of the tuberculosis. In two other patients, one of whom had had an intrapleural and the other an extrapleural pneumonectomy, it was necessary to remove the sponges. One of these patients ran an elevated temperature for from six to eight weeks, presumably from an empyema. The sponge was removed and the pleural cavity drained. Through the drainage tube and the use of antibiotics, streptokinase and streptodornase, the empyema cavity was sterilized and the tube was removed. At a later date, Lucite balls were placed in pleural cavity with a very satisfactory result. The other patient developed a discharging sinus and we waited about six weeks, hoping the sinus would close. When the sinus failed to close, the sponge was removed and the empyema cavity was drained and sterilized, much as in the case of the other patient who had had a pneumonectomy. Subsequently, Lucite balls were used with a satisfactory result. One other patient, who had a sponge prosthesis following a right lower lobectomy for suppurative bronchiectasis and multiple small lung abscesses, developed an empyema and now, six weeks later, still has the drainage tube. Possibly the sponge should not have been used in this patient, but she was a very pretty girl and anxious not to have any structural deformity. Possibly sterilization of the sponge was not as effective as it should have been.

It is readily apparent that, based upon my personal experience with the sponge, with four complications out of 25 cases, I am somewhat cautious about its use. When the sponge is satisfactory, the results are beautiful. However, an infection with a sponge is of a more serious nature than an infection without the sponge. In my considered opinion, this particular sponge may not be the ideal prosthesis for this reason. I hope, however, that this report of Dr. Gale and Dr. Curren will stimulate the interest of other men and that the sponge or other prosthetic material will be given a further trial.

DR. ROBERT T. FOX, Chicago, Ill.—I enjoyed very much this paper by Dr. Gale and Dr. Curren. In Chicago, we too are afraid of overexpansion of the remaining pulmonary tissue especially, as Dr. Gale has emphasized, when the resected lobe or segments are fairly large. The problems of tailoring thoracoplasty have of course been discussed before. We have been reluctant to do it at the time of resection, but consistent with our technique as described by Dr. Adams last year, we have turned again to the paraffin pack in the subscapular space, doing a limited thoracoplasty at the time of pulmonary resection in cases where we feel we want such a collapse. Unfortunately, many of these resections require major extrapleural dissection, and we have been reluctant to use prostheses here, even in the subscapular space, because of the very thin layer separating the pleural and subscapular space in such cases. But where we have a good parietal pleura we have been using such a pack, and I should like to show one case to illustrate this procedure.

(Slide) This is a patient about 50 years of age. You see the cavity high on the left side. He was in the sanitarium just a short time and was dissatisfied with his treatment for some reason or other and left, and a pneumothorax was started on the outside.

(Slide) Again he failed to convert to negative and continued to have cough and expectoration, and was readmitted. You see the pneumothorax here and the cavity still open, practically two centimeters in diameter in the partly collapsed upper lobe. The pneumothorax was abandoned but the lung failed to expand completely, and resection was decided upon. We did a thoracotomy and found that there was considerably more nodular disease throughout the upper lobe than would meet the eye on various roentgenograms. After resecting the upper lobe and decorticating the lower lobe, there was still evidence that the lower lobe would not fill the space satisfactorily.

(Slide) After closing the rib cage, the second, third, and fourth ribs were resected, and a small paraffin pack of about 250 Gm. was inserted. This patient had a very ex-

ed at postoperative course. There was no paradoxical motion of the chest wall as there would be without such a prosthesis, and we feel that this is an excellent procedure in these cases. Where extrapleural dissection is necessary, we are looking forward to trying the Ivaleon prosthesis, and hope that our results will be as satisfactory as Dr Gale's.

DR O THERON CLAGETT, Rochester, Minn.—That surgeons are not entirely satisfied with thoracoplasty as a surgical procedure is evidenced by the variety of techniques that have been devised to accomplish the purposes of thoracoplasty without the deformity that results from it. I believe that Dr Grindlay at the Mayo Clinic was the first to use the plastic sponge that Dr Gale has described. About four years have elapsed since these sponges were first used in the pleural space following pneumonectomy performed in dogs. We have observed no untoward effect from the use of this sponge in the pleural space of experimental animals. The sponge prevents mediastinal shift, it causes no tissue reaction of consequence. As Dr Gale has described, fibroblasts grow out into the meshes of the sponge but it has been possible to remove the sponges without difficulty. Our clinical experience is much less extensive than Dr Gale's. However, we do have one patient in which the plastic sponge has been in the pleural space for about three years and several other cases in which the sponge has been in place for shorter periods. We have had no complications that could be blamed on the sponge itself. We have placed the sponge both intra- and extrapleurally. It is too early for accurate evaluation of the usefulness of this material. We have had disappointments with other foreign bodies that have been used in human beings in the past. We may be disappointed with this material in the future. However, our experience to date is most encouraging. We believe with Dr Gale that the procedure he has described is worthy of further trial.

DR SVEN LUNDBERG, Goteburg, Sweden.—I wish to thank you for the privilege of attending your Association. Dr Gale's lecture has been very interesting, and I have applied myself with the same problems. In the surgery of pulmonary tuberculosis the question before and after operation is more and more respected. In earlier times lung resections were done, not always accompanied by plastic operations. In many of these cases we can find a distention of the remaining lung tissue with emphysema that causes a loss of function considerably greater than the diminution of function owing to the resection. In order to maintain the best function of the remaining lung tissue, I think it is necessary to reduce the size of the thoracic cavity by thoracoplasty before, at the same time, or after resection. Thoracoplasty before resection causes technical difficulties for the second operation, the simultaneous operation increases the risk of a plastic operation after resection is to be preferred, but it is not good for the patient to wait for a subsequent operation.

and in the lower part the prosthesis with some exudate and displacement of the heart to the left. (Slide) Two months after resection the heart is in normal position and (Slide) three months after resection the heart is still in normal position

(Slide) This is a case of pneumothorax with the remaining cavity. The picture was taken some days after resection and again you see displacement of the heart to the right. (Slide) One month later there is exudate and we can see the sponge in the upper part. The heart is in normal position and (Slide) this was taken two months after resection

(Slide) You see in this case a great cavity in the right upper lobe. I did a six rib plastic, but there was a remaining cavity at the apex. (Slide) Then I performed a resection of the right upper lobe and put in a prosthesis and, as you see, below that there is exudate, but I hope that after some months the exudate will be resorbed and that we will have normal function of the lower part of the lung

DR GALE (Closing) —I wish to thank those who were kind enough to discuss this paper. I do not want to leave the wrong impression. Certainly one would be rather foolish to claim too much for a sponge such as this after using it in only 50 cases. It is a foreign body. Many of you who started using paraffin years ago know how tricky it was, as well as any prosthetic appliance of any kind.

I think we must continue to feel our way, and if there is any doubt as to the presence of contamination of the intra- or extrapleural space at the time of operation, please do not insert the sponge. I also feel that as time goes on and more experience is gained, we will know just how safe it is. It is not the type of procedure that can be carried out by everyone or on every case. Each patient will have to be evaluated separately.

CIRCULATORY ARREST

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CIRCULATORY arrest after local anesthesia or during operation has such a high mortality that the generally accepted principles of treatment must be revised.

ETIOLOGY

The cause of circulatory arrest is in most cases an anoxia and the best treatment is prevention. In cases with local anesthesia idiosyncrasy to the drug or injection in a vessel may be the cause.

PATHOPHYSIOLOGY

During operations with the chest open the development of a circulatory arrest has been observed several times. In most cases the heart rate is rapidly falling down to 10 to 20 beats per minute. A standstill of the heart then comes in a very short time. In very few cases the circulatory arrest is preceded by a gradual increase in the heart rate, an increasing number of extrasystoles and finally ventricular fibrillation. The heart may also come into a ventricular fibrillation secondary to a standstill, especially during a delayed resuscitation.

Differential diagnosis between a heart standstill and a ventricular fibrillation can in a short time, be made only by direct inspection of the heart in the catastrophic cases. If a direct writing electrocardiograph is connected, a ventricular fibrillation is demonstrated, but in standstill atypical and misleading tracings may be produced.

TREATMENT

Theoretical Considerations—The treatment is entirely different in the two types. Injection of Adrenalin is indicated in cases of heart standstill but contraindicated in ventricular fibrillation where the only effective treatment is electric shock. Therefore the logical treatment which is now generally accepted is: (1) opening of the chest and the pericardium for diagnosis and treatment; (2) artificial positive pressure ventilation through an endotracheal tube; (3) injection of Adrenalin into the heart in the case of a heart standstill; (4) injection of Adrenalin into the heart in the case of a heart standstill; (5) electric shock into the heart in case of ventricular fibrillation; and (6) continuation of resuscitation and if necessary repetition of the measures mentioned under (1) to (5).

PRACTICAL CONSIDERATIONS

A dog's heart may be without oxygenated blood for 30 minutes and then restart its regular rhythm with complete survival provided brain perfusion with oxygenated blood is performed. If the human brain however is without

oxygenated blood for more than 3 to 4 minutes, irreparable changes will occur. Therefore two requirements have to be fulfilled to permit the above mentioned scheme: (1) the chest must be opened and the heart massage instituted within 3 minutes from the time the diagnosis is made, allowing a maximum of one minute to establish the diagnosis, and (2) a defibrillator must be at hand for the electric shock. If these two requirements are not fulfilled the result will only be a temporary restitution of normal cardiac action in a decerebrated patient.

The above mentioned scheme should then be followed routinely in all cases with an open chest during operation and in most cases during an intervention in the upper abdomen. In all other cases of circulatory arrest the author follows a more conservative plan.

1 Adrenalin, 1 1000, and a syringe with a long, widebore needle is always at hand. As soon as the diagnosis is established the needle is inserted into the heart 2 fingers breadth lateral to the sternum between the fourth and fifth ribs on the left side. A widebore needle enables the surgeon to quickly aspirate blood and thereby make sure the needle is accurately placed in one of the chambers of the heart.

2 One milliliter of Adrenalin, 1 1000, is injected. If this is done quickly enough and the heart is still beating slowly, an immediate response is noted, the pulse returns and the needle, which moves with the heart beat, can then be removed.

3 If, a few seconds after the first injection, no response is noted 5 ml Adrenalin, 1 1000, is injected. An immediate response may be noted in most instances. If not, 10 ml Adrenalin 1 1000 is given.*

During these injections only one minute is lost. In the meantime artificial positive pressure ventilation is instituted through an intratracheal tube.

Following this scheme it has been possible to re-establish a regular cardiac beat in 4 successive cases. In two cases the recovery was complete, in the other two an immediate response to 5 ml Adrenalin, 1 1000, was achieved, but the injections were made too late and cerebral damage had already occurred.

The value of intracardiac Adrenalin injections in cardiac resuscitation has been underestimated. Physiologists have warned us and described the bad reaction to Adrenalin of the beating heart in experimental animals. An intravenous injection of 0.5 ml of Adrenalin 1 1000, made by mistake on a healthy 20-year-old girl was lethal†. Most surgeons use only 1 ml of Adrenalin, 1 1000, which is too little and is often without effect.

Two such cases with circulatory arrest after local anesthesia for thoracoplasty were experienced in 1949.

* A combination of Adrenalin and 5 ml 1 per cent procaine may be valuable. As, however speed is the over all important thing the Adrenalin alone is preferred if some seconds are gained by omitting the procaine.

† On the other hand Crafoord during an intrathoracic operation by mistake injected 10 ml Adrenalin 1 1000 in the heart at standstill. There was an immediate response with complete recovery and no untoward effects.

The heart in standstill is anoxic, the reaction delayed and a big initial dose is needed. No untoward effect is noted once the heart is beating again as the action of Adrenalin is too short.

CASE REPORTS

CASE 1—July 21, 1949, a 41 year old man had local anesthesia with paravertebral block, brachial plexus block, and infiltration in the line or incision or the subcutaneous and muscle planes (0.5 per cent procaine with tetracaine 0.5 1000). About 3 to 4 minutes after the completion of the anesthesia, the patient's pulse suddenly stopped. Artificial ventilation through an intratracheal tube and 1 ml Adrenalin, 1 1000, in the heart had no effect. The patient died. Post mortem examination showed right sided pulmonary tuberculosis.

CASE 2—November 8, 1949, a 27 year old woman received 230 ml of 0.5 per cent procaine with tetracaine 0.5 1000 in local anesthesia with paravertebral block, brachial plexus block, and local infiltrations in the subcutaneous and muscular planes. Fifteen minutes after completion of the local anesthesia and during performance of the incision, the pulse rate suddenly increased but after a short interval the pulse could no longer be felt. Intracardiac injection of 1 ml of Adrenalin 1 1000, and artificial ventilation through an intratracheal tube was without effect. Post mortem examination revealed nothing other than bilateral pulmonary tuberculosis with a big cavity in the right upper lobe.

With this experience of two deaths following circulatory arrest after local anesthesia treated by intracardiac injection of 1 ml Adrenalin, 1 1000 and artificial ventilation, I decided, in a similar situation, to follow the advice to perform an immediate thoracotomy with heart massage. It is, however, easier to make the decision than to carry out the treatment if the surgeon is operating in a sanatorium which is equipped only for thoracoplasties made under local anesthesia which are performed by the surgeon himself.

CASE 3—Two years later this case of circulatory arrest occurred after epidural anesthesia with 9 ml Xylocain 1 per cent for a second stage thoracoplasty. The patient was a 57 year old man with a right sided tuberculous empyema, who 25 days earlier had had a first stage 5 rib thoracoplasty with apicectomy. It was clear that the general accepted treatment with cardiac massage was impossible. The right lung was too small with an atelectatic upper lobe and a big empyema to take over the function alone. After a left sided thoracotomy for cardiac massage. A right sided thoracotomy was performed in 3 minutes (see roentgenogram, Fig. 1). It had been easy for a surgeon to open the left chest. The outcome of such a treatment with cardiac massage will, however, most probably be fatal. For that reason the following treatment was chosen: (1) Immediate intracardiac injection of 2 ml Adrenalin 1 1000, without respiration. (2) The surgeon introduced an intratracheal tube and gave artificial ventilation at blowing his own expiration air into the tube during the time the anesthesia was connected. (3) After 3 to 4 minutes of cardiac arrest 5 ml of Adrenalin 1 1000 was injected into the heart with immediate return of the pulse which could be felt. (4) Then another cardiac arrest occurred. After immediate intracardiac injection of 10 ml of Adrenalin 1 1000 regular heart beat reappeared. On half an hour spontaneous breathing began and after 7 hours the intratracheal tube could be removed. After 2 days he had recovered completely but had no memory of the 2 days.

In all 20 ml of Adrenalin 1 1000 was given intracardially which saved the patient's life. With this experience I cannot agree with Ehrenkrantz and Sjöström (1951) who make the conclusion that "epinephrine must be used most often or not at all".

In cardiac arrest a much larger dose of Adrenalin in the heart is needed to start beating. The reaction of the anoxic heart to Adrenalin is very weak. Thus a high initial dose is needed. The action of Adrenalin starts to run into effect once the heart is beating again.

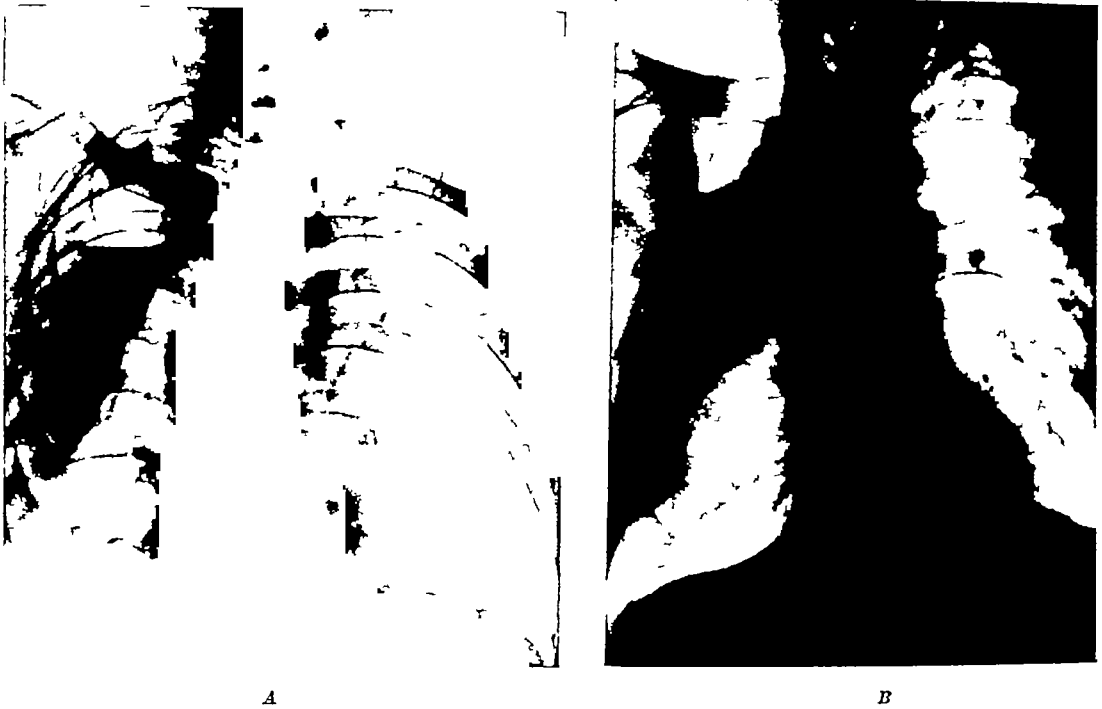


Fig 1—A, Case 3 a 57-year-old man. Roentgenogram taken on July 13 1951 showed right sided thick-walled tuberculous empyema. On July 21 1951 a 5 rib thoracoplasty with apicolysis was performed. B, Case 3. This was the roentgenographic appearance when the circulatory arrest occurred. Twelve days later still a small left sided pneumothorax remained after the three intracardiac injections.



Fig 2—A—Case 4 a 51 year old woman. Roentgenogram taken on Aug 17 1951 showing extensive left sided tuberculosis fixation of the left diaphragm, and a thickened left pleura.



Fig. 1. Chest X-ray taken on Dec. 29, 1951, three weeks after a first stage 3 rib thoracoplasty with plecolysis.



Fig. 2. Chest X-ray taken on Dec. 29, 1951, three weeks after a first stage 3 rib thoracoplasty with plecolysis.

If the heart, however, is beating slowly, no pulse being palpable, the smaller dose of 1 ml may be sufficient and should therefore always be given as the first injection. If no immediate response to that dose is noted, 5 ml and, if necessary, 10 ml Adrenalin, 1 1000, should then be injected with the needle left in place in the heart to save time.

This plan has then been followed in all cases except those in which, due to the nature of the surgical procedure, the direct cardiac massage is a quicker procedure.

CASE 4—A 51 year old woman with extensive left sided tuberculosis had a first stage thoracoplasty with removal of the upper 3 ribs and apicolysis done Dec 7, 1951. Four weeks later a second stage with removal of posterior parts of ribs 4, 5, 6, 7, and 8 was completed (see roentgenogram, Fig 2). The second stage was done under epidural anesthesia with 50 ml 1 per cent Xylocain. Sixty minutes after completion of the anesthesia, when the operation was finished, there was a fall in blood pressure. No blood was available for an immediate transfusion. In spite of intravenous nor adrenalin the blood pressure continued to fall rapidly until no pulse could be felt and the patient stopped breathing. (1) She was immediately turned on her back, (2) 6 ml. Adrenalin, 1 1000, was injected intracardially through the left fourth intercostal space, (3) there was an immediate response with return of the heart action and a blood pressure of 160, (4) the patient was still bluish and without spontaneous breathing so artificial ventilation was instituted through an intratracheal tube, (5) after about 1 hour spontaneous breathing returned, and (6) after 1½ hours the patient had regained consciousness. The aftercourse was uneventful.

This 51-year-old woman had extensive tuberculosis in her left lung, diaphragm fixation, and thickened pleura after a left pleumisy. When the circulatory arrest occurred after the second stage thoracoplasty, the chest surgeon did not judge it to be possible for him to open this left chest and perform cardiac massage within 3 minutes. Experience from extrapleural resections for tuberculosis under thoracoplasties has shown these incisions to be more time consuming. A right-sided thoracotomy with cardiac massage would not have been tolerated from a functional point of view (see roentgenogram, Fig 2C).

In two more cases the efficiency of a big dose of Adrenalin intracardially to overcome circulatory arrest has been found.

CASE 5—A 39 year old woman with a residual tuberculous cavity under a 5 rib thoracoplasty underwent a right upper lobectomy with resection of the apical subsegment of lower lobe. The endobronchial anesthesia technique had been used with nitrous oxide, oxygen, and curare. At the end of the operation, when the skin was being sutured, 1.5 mg Prostigmin and 0.5 mg atropine was given. Two minutes later a copious amount of clear yellow fluid was pouring out from the lungs. The patient was turned over on her back and aspiration was carried out through the tube. The surgeon had not experienced such an idiosyncrasy to Prostigmin before and instead of removing the tube and sucking out the bronchial tree through a bronchoscope he continued an ineffective suction through a small plastic catheter.

Circulatory arrest due to anoxia occurred. There was, however, a delay of more than 3 minutes before 5 ml Adrenalin, 1 1000, was injected intracardially with an immediate return of heart action. Three and one half hours later spontaneous breathing returned. Six hours after the operation the patient died without having regained consciousness. Post mortem examination showed pulmonary edema but no hypertrophy of the right ventricle. Death was due to the anoxic brain damage.

CASE 6—A 20 year old man had a 5 rib thoracoplasty performed on the right side. A hematoma developed in the apicolysis space compressing the right lung. One day after the operation the wound was reopened and the hematoma evacuated in local anesthetic. At the end of the procedure, circulatory arrest occurred. The right lung was inflated. Adrenalin and a suitable needle was not at hand until more than 4 minutes had elapsed. Five milliliters Adrenalin, 1:1000, was given intracardially with an immediate return of heart action. The patient died 5 hours later without having regained consciousness. Post mortem examination did not show the origin of the bleeding.

SUMMARY

In cases of circulatory arrest the generally accepted treatment with thoracotomy and cardiac massage cannot be adopted as the method of choice in all types of cases.

- 1 The best method is always prevention when possible.
- 2 In many bilateral tuberculous cases the pathologic changes of lungs and pleura will make it impossible for a chest surgeon to open the thorax and perform cardiac massage within 3 minutes.
- 3 Due to the diminished respiratory reserve many tuberculous patients do not tolerate an unexpected thoracotomy.
- 4 In two such cases the author administered large doses of Adrenalin intracardially with survival of the patients. None had lasting neurologic symptoms.
- 5 In circulatory arrest the author now immediately injects 1 ml. Adrenalin 1:1000 through a wide bore needle into the heart. If no immediate return of heart action is found, 5 ml. Adrenalin 1:1000 is injected through the needle left in place in the heart. This dose has so far been sufficient in most cases. If no pulse is palpable after one minute an additional 10 ml. of Adrenalin 1:1000 is injected.
- 6 If this treatment is instituted later than 3 minutes after the circulatory arrest, the result is most often a return of heart action in a patient with cerebral damage.
- 7 The author believes that direct heart massage should only be performed in cases where, due to the nature of the operation, this is the faster procedure.
- 8 As time is the most important factor in circulatory arrest the author believes that the most rapid procedure should be chosen. It is thought that many patients are saved by immediate intracardiac injection of large doses of Adrenalin compared to the number lost by cardiac massage one minute later or due to ventricular fibrillation.

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ATELECTASIS PHYSIOPATHOLOGY AND TREATMENT

SPECIAL REFERENCE TO ITS OCCURRENCE IN BULBAR POLIOMYELITIS, AND TO A
SUGGESTED THERAPEUTIC AID BY THE INTRABRONCHIAL ADMINISTRATION OF
TRYPSIN

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ATELECTASIS has long been recognized as a pathologic process. Traube, in 1846, was able to collapse a lung or lobes of a lung by direct mechanical obstruction of a bronchus through creation of an artificial plug with shot or gum arabic.¹ The writings of Pasteur² describe numerous cases which resulted from paralysis of the diaphragm during diphtheritic infection. Other reports have appeared in the literature, but it is only within very recent years that practical application of this information has led to more enlightened methods of therapy. Jackson³ in 1925 was the first to correlate the clinical and experimental findings and successfully to cure an atelectasis by bronchoscopic aspiration of a diphtheritic membrane. Haight⁴, twelve years later, stressed the significance of atelectasis as an etiologic factor in postoperative bronchopneumonia. During the last war particular progress was made toward the general awareness of this problem through the experience of Burdard, Burbank, Sampson⁵ and others with the traumatic wet lung.

The recognition of atelectasis as a significant complication in poliomyelitis is also fairly recent. Sutherland⁶ in 1913 was the first to mention collapse of the lung among the complications of this disease. Regan,¹² eleven years later, again reported instances of this occurrence. Subsequently, only years elapsed before further reference was made to this pathologic process. At this time, Cooperstock⁷ discussed the condition and recognized it as a definite complication of poliomyelitis. Perhaps the late cognizance of this complication is due to the fact that improved care over the last decade has considerably decreased the immediate mortality from bulbar poliomyelitis. Previously, the great majority of these patients did not live long enough to develop atelectasis. Since the care of these patients is still improving, we anticipate that the problem of atelectasis in respiratory poliomyelitis will continue to increase.

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It would seem worth while, therefore, to discuss the etiology of atelectasis, and to emphasize particularly the problem as it presents in patients with bulbar poliomyelitis

PATHOPHYSIOLOGY

Three major requisites are necessary for development of atelectasis in a lung, lobe, or segment. First, some mechanical cause for obstruction must be present. Second, there must be a sufficient circulation to allow absorption of the air distal to the obstruction. Third, there must be no accessory means for an entry into the distal portion of the obstructed area.

Normally, the body guards against the occurrence of atelectasis by several physiological mechanisms. Alteration in any one of these factors may contribute to its occurrence or may influence the severity of resulting signs and symptoms. These factors may be enumerated as follows: (1) efficient ciliary action on a normal bronchial mucous membrane, (2) a normal exudate from the bronchial mucosa, (3) an efficient collateral respiration, (4) a normally functioning process for disposal of secretions once they reach the pharynx, (5) an efficient cough mechanism, (6) absence of infection in the tracheobronchial tree.

Normally, secretions are removed by continuous ciliary activity, together with the expulsive action of bronchial inspiratory and expiratory movement. The ciliary action begins proximal to the respiratory bronchioles but in the presence of allergy, chronic irritation, chronic infection and asthma, a metaplasia of the ciliated epithelium may occur,¹⁴ thereby eliminating normal ciliary activity. Under such circumstances abnormal or excessive secretions are aided in their removal by the cough mechanism and by lymphatic absorption.

The effectiveness of ciliary action depends to a large extent on the viscosity of the secretions. The bronchial glands secrete both mucin and serous fluid. Buhrmester⁵ has demonstrated that there is a direct relationship between the mucin content and the viscosity of secretions. In the presence of chronic irritation, inflammation, or allergy, there is an increased production of mucin with consequent increase in viscosity and adhesiveness of the secretions. Dehydration also will lead to an increased mucin content. Similarly, the type of infecting organism present in the secretions is a consideration in some instances. For example, pneumococcal sputum is extremely viscous.³ It is also worth recognizing that sputum coughed up is not necessarily an index of the type of bronchial mucus or exudate occluding a segment or lung. It may rather represent an overflow from the mouth of the bronchus of the affected lobe, even though the bronchus itself is completely occluded. Further, the concept of obstruction of the large bronchi by discrete, essentially solid plugs is rarely found in actual practice. Instead, the occlusion of small bronchi and bronchioles by viscid secretions is more generally seen.

The third factor in this series of mechanisms is an efficient collateral respiration. Otherwise one would expect segmental or subsegmental atelectasis to occur more commonly. This is especially true in poliomyelitis, where

large and small bronchi are often filled with mucus which may be present one time before being suctioned off, it removed at all. Moreover, if this were not the situation, one would expect every bronchitis to lead to a spreading patchy atelectasis by the plugging of the smaller bronchi thereby preventing the passage of air to the distal portions of the segment. Generally, an distal to the obstruction can assist in the expulsion of mucus plugs in the act of coughing, thus preventing eventual suffocation.¹³

The existence of collateral respiration was discovered by Van Allen in 1930 in association with Adams and Lindskog.^{14, 21} It implies that the pulmonary lobules are not end organs from the standpoint of ventilation.¹ It has been demonstrated that after the direct bronchial route for air is obstructed artificially, there still exists an indirect and peripheral route for exchange and maintenance of aeration from adjacent ventilated segments. However, obstruction of a main bronchus would prevent air from entering in entire lobe, and therefore such collateral ventilation would not occur. Since collateral respiration is not dependent on collateral circulation it tends to lessen the incidence of obstructive atelectasis, equalize pressure changes in the lung, equalize alveolar gas tension of O_2 and CO_2 and provide a more uniform oxygenation of venous blood. There is some evidence to show that the pores of Kohn, small anatomic defects in the alveolar walls, may provide the route for such exchange.

The cough mechanism is in itself dependent on many factors. First, the ability of the respiratory muscles to function; second, the presence of mucus in the lung tissue; third, the ability of the glottis to open and close; and fourth, the presence of a normal central cough center, a function that is not well understood.

Humbeker,²² in 1927, showed clearly that during respiration the bronchi change their caliber, being larger during inspiration and smaller during expiration. Recently Di Rienzo¹ has confirmed these observations. From these studies it has been conceived that a cough is a dynamic act of the mucous membrane that expels mucus secretions by means of a high speed peristaltic wave originating in the small bronchi and ending in the larynx. This is accompanied by the harmonic movement of the functional sphincters. This peristaltic bronchial wave may in itself move along secretions, but the greater the contraction distal to the secretions, the more effective will the cough be in moving any obstruction. Again, abnormal pulmonary conditions such as emphysema or bronchiectasis will cause excessive accumulation of secretions and interference with these dynamic bronchial properties.

In poliomyelitis, the cough mechanism may be impaired either centrally or peripherally by paralysis of the muscles involved, such as the diaphragm, intercostal and intercostal muscles. Also, paralysis of the larynx will prevent abduction and adduction of the cords necessary for the forced expiration of intrathoracic and intrapulmonary pressure required for the coughing reflex. Poor bronchial drainage from the hyperinflated and overdistended lungs in respiratory poliomyelitis cases will also contribute to the

and mobility of the bronchi. Also, a reduction in vital capacity will vitiate against an effective cough.⁸

A defective or absent cough in the poliomyelitic patient is usually accompanied by difficulty in disposing of secretions once they have reached the pharynx or throat, and these cases require prompt suction for the removal of these secretions. Similarly, in those patients with tracheostomies, any delay in aspiration from the trachea may lead to further stagnation of the secretions with consequent increase in viscosity, or to subsequent reinhalation of the secretions into distal bronchi or bronchioles.

The obstructing agent in poliomyelitis is either from bronchial secretions or from aspirated exogenous material. In either case, this material is infected and the severity of the atelectasis will to a great extent be dependent upon the degree of infection present. Coryllos⁹ placed a sterile obstructing balloon into the bronchi of dogs and left it there for sixteen to twenty-four hours. During this period atelectasis ensued, with slight fever, increased respiration, cough, and typical radiographic findings. After removal of the balloon, gradual re-aeration in from sixteen to twenty-four hours took place. When the bronchus was obstructed with an infected balloon or with plugs of sputum containing bacteria of varying virulence, it was noted that the severity of symptoms as well as the mortality varied with the virulence of the organisms. Moreover, the infection seemed to spread from the site of obstruction through the bronchial lymphatics and into the distal lung parenchyma, setting up a consolidated segment which could lead to abscess formation or even gangrene. If infection set in, the possibility of re-aeration became less, even though the primary obstructing factor was removed. It is accepted, however, that a variable amount of alveolar exudate is absorbed from the lungs over a period of time. Head¹³ states that when the parenchyma distal to an obstructed segment or lobule becomes opaque on x-ray film, it does so because of consolidation and not primarily because of airlessness. In poliomyelitis, atelectasis often follows infections of the upper respiratory tract, and the implication of this as far as the application of therapy is obvious. Again the atelectasis may be slow or rapid, with or without obvious signs and symptoms (see Case 2).

The length of time a segment of lung may remain collapsed, and yet still be re-aerated, is a debatable point, but in general depends on the presence and degree of infection. Under vigorous antibiotic therapy, infection to all intents and purposes may be eliminated, and with the removal of the obstructing agent, complete reabsorption of the parenchymatous exudate may take place within a short time. As early as 1903 Ascoli and associates¹ and later Bittori⁴ observed that liquefaction of parenchymatous exudate was greatly aided by the liberation of two proteolytic enzymes from the white blood cells in the exudate. If infection occurs in the distal segment, irreversible pathologic changes may appear within a short while. If infection has not occurred, and it is often difficult to determine this, re-expansion may be obtained after many months, if not longer.

THERAPY

The understanding of the above factors in the etiology and pathogenesis of atelectasis allows formulation of a reasonable approach to therapy. Generally, the fault does not lie with a single factor but more often is due to a combination of several or all of them. It is the rare case where one can remove a single obstruction or plug, and following this have the lung return to normal. Such an example is the removal of a foreign body soon after its aspiration. In respiratory poliomyelitis cases there may be a combination of diminished respiratory function, absent or defective cough inability to dispose of pharyngeal secretions, abnormal bronchial secretions in quantity and quality plus respiratory infection. One sometimes finds that these patients may also have had asthma or chronic bronchitis, which has led to a damaged bronchial mucous membrane. In such cases there is generally need for more than simple aspiration of the larger bronchi, although rarely spontaneous recovery without any specific therapy may occur. Cooperstock reports that three of his four cases cleared on dimethylamide therapy alone. We have had one instance in which re-expansion occurred five months after development of a right upper lobe atelectasis.

Many methods have been described for the prophylaxis of this condition deep breathing exercises, inhalation of high concentration of CO_2 and intravenous sodium therapy, but these methods are useful in short term therapy such as postoperative states. In general they are impracticable in the chronic poliomyelitis. In the respiratory poliomyelitis intermittent positive pressure therapy and the oscillating bed have been advocated but proof of their effectiveness is not convincing.

The management of the patient who has developed atelectasis should be directed at satisfying the following conditions: (1) removal of the obstructing agent, (2) loosening and removal of viscid bronchial secretions, (3) establishment of an efficient collateral respiration, (4) aid in the removal of the viscid bronchial and pharyngeal secretions, (5) assistance in the securing of a good cough, (6) treatment of any incipient infection.

Bronchoscopic aspiration as indicated above does not necessarily remove the secretions in the smaller bronchi and bronchioles but by opening up the larger proximal and segmental branches allows collateral respiration to be established. Once suction of these bronchi has been performed insufflation of collapsed lung with oxygen under slight pressure through the bronchoscope and in getting some air into the distal obstructed areas.

Atelectasis of long standing may be aided considerably by loosening of the adhesions. Baker and associates advocate the use of iodides which have been demonstrated experimentally will increase the amount of serous exudate direct stimulation of the bronchial glands or by the alteration of the permeability of the bronchial mucosa. The injected 1 to 2 Gm of 1% sodium iodide intravenously and noted by means of bronchoscopy a stimulation of secretions occurring more to little in minutes and lasting two to three days and producing on the amount of sputum collected. The treated 11 cases of chronic atelectasis were all received pre- and post-operative

antibiotics with 1 to 2 Gm of sodium iodide intravenously twice a day for 3 to 4 days after onset of the atelectasis. These patients were ambulated early and encouraged to cough. By this means the condition cleared within twenty-four to forty-eight hours. Generally, in the poliomyelitic patient this therapy is not advisable, as it only will increase the amount of secretion with which the patient is already having difficulty. The same would apply to aerosol therapy with antispasmodics, which Christie and co-workers⁷ have found so effective in their cases. They report that the aerosol inhalation of 1 per cent epinephrine and 1 per cent ephedrine (in a proportion of 1:9), or Neo-Synephrine five to eight times a day, releases bronchospasm. The patient, if then given an expectorant mixture to loosen up secretions, can often rid himself of all secretions and relieve the atelectasis.

Recently there has been considerable interest in the use of various enzymatic agents for physiologic débridement. There are currently available three such products: Tryptai,* Varidase,† and Caroid‡. Muenster and associates¹⁷ have recently reported on the successful intrabronchial instillation of streptokinase and streptodornase in a long-standing case of atelectasis. There was no clearing of a left lower lobe atelectasis following three bronchoscopies, so one month later they instilled 20,000 units of streptokinase plus 9,500 units of streptodornase in 5 cc of solution into the left lower lobe bronchus. The patient responded by coughing up thin watery secretions. The procedure was repeated the following day with double the above dosages, and, on bronchoscopy, it was noted that there was no mucosal change. There was a slight elevation of temperature, and on the third day there was clinical and radiological improvement following the coughing up of these secretions.

The product Tryptai has attracted our attention for two reasons, (1) it is an enzyme occurring normally in the human body, and (2) there is within normally living human cells an antitrypsinoginase which effectively prevents its action except on dead or injured tissues. Investigation has also demonstrated that mucoid material from man is effectively dissolved by the action of trypsin. Such physiologic activity seemed to us to justify the application of purified crystalline trypsin to the management of certain difficult cases of chronic atelectasis unrelieved by the usual methods of therapy. Ideal candidates for such a study are those patients with severe bulbar poliomyelitis who secondarily develop persistent atelectasis.

Roettig and associates²⁰ reported on the use of trypsin in chest diseases and noted that other than a histamine like reaction which occurred with the intrapleural injection of this substance in cases of empyema, and which could be prevented by adequate antihistaminic therapy, the procedure was relatively safe. They also found that in tuberculosis empyemata, the number of tubercle bacilli rapidly decreased. It has been postulated that this may be

*Armour Laboratory's preparation of purified crystalline trypsin. Grateful acknowledgment is made to this organization for supplying this material as used in the clinical and experimental observations.

†Eliel Laboratory's preparation of streptokinase, streptodornase.

‡American Ferment Company's preparation of the vegetable enzymes papaya and papayatin obtained by fermentation from Carica Papaya.

to the action of trypsin on the tubercle capsule thereby making the organism susceptible to ordinary antibiotics.¹¹ Roetting treated 10 cases by aerosol trypsin, giving 100 mg. in 2 c.c. of Sorensen's Phosphate Buffer solution each day. He noted a decrease in the viscosity of secretions and sputum but these cases did develop a slight irritation of the tracheobronchial tree as well as hoarseness. We have given trypsin endobronchially to four patients with atelectasis two of whom had poliomyelitis. These poliomyelitic patients were carefully selected because of the severity of their basic pathology and because their prognosis seemed so poor without the aid of something more definitive being accomplished, bronchoscopy alone having failed to remove the obstruction. In most instances therapy was carried out while the patient was in the Monaghan respirator. Initially small doses of Trypsin were utilized but as confidence in the procedure increased a standard dilution was established. This consisted of 250 mg. of trypsin in 5 c.c. of Sorensen's Phosphate Buffer solution.

The procedure used was to place the patient in a semireclining position at about 20 degrees and propped it about 30 degrees with the affected side down. The bronchoscope was then introduced and all endobronchial secretions aspirated from both main stem bronchi and all orifices. Subsequently the bronchoscope was passed into the main stem bronchus of the dependent side and trypsin solution was slowly introduced through the branch oxygen inlet of the bronchoscope. By slow introduction the trypsin was aspirated into all potential bronchial orifices. The scope was then removed and the patient allowed to remain in the same position for one hour. The bronchoscope was again introduced and all secretions completely aspirated.

Bronchoscopy in our poliomyelitic patients has not required anesthesia since they all have had tracheostomies.

One of the major problems in the respiratory poliomyelitic invalid is the inability to cough efficiently in order to bring up any secretions. Barish and Gentes describe the use of an exsufflator which can be adapted to the tank of a respirator or to a pressure hood from which a high volume flow rate of air during expiration can be achieved and which would be equivalent to a moderately vigorous cough. This is done by the gradual withdrawal of air until a negative pressure of about 40 mm. Hg is obtained and then suddenly raising the pressure so that it rises to atmospheric pressure within 1.004

l. The rate is set from six to eight times a minute to prevent hyperinflation with the pressures used and to prevent impairment of cardiac output.

This high degree of inflation of the lung produced by 40 mm. Hg negative pressure has the added effect of increasing the diameter of bronchi and facilitating the passage of air beyond the obstructing mucus or tracheal casts, thus increasing the effectiveness of the expiratory blast in pushing secretions into the larger bronchi, trachea and pharynx. It is suggested that five courses of treatment of one half hour each be given.

It is required to accomplish complete clearance of the lungs and to prevent atelectasis. The procedure is stopped at ten minutes if the patient is unable to tolerate it.

It is suggested that the procedure be

Perhaps the use of this exsufflator following endobronchial instillation of trypsin would aid greatly in the handling of cases of long standing atelectasis.

Finally, one must control infection. It has been our policy to treat any upper or lower respiratory tract infection in our poliomyelitic cases vigorously with antibiotics, and in the eventuality of a suspected or proved atelectasis to give large doses of penicillin. If throat or laryngeal cultures demonstrate organisms that may be more sensitive to other antibiotics, these may also be used.

The suctioning and aid to the patient in the removal of secretions from the upper respiratory tract is part and parcel of normal nursing, but, following attempts to relieve an atelectasis as described above, particularly after the instillation of trypsin or other drugs which loosen or stimulate secretions, special attention and repeated suctioning for some time will be necessary.

Following the bronchoscopic aspiration and other therapy advocated, we feel that the encouragement of deep breathing either by means of strong respiratory pressures, or, better still, by means of a positive pressure apparatus for ten to thirty minutes three to five times a day is useful, particularly if one precedes this by prior antispasmodic aerosol therapy. In our wards we have found Isypnel, five drops in 20 drops saline given as an aerosol through the oxygen tubing, particularly useful. The exsufflator, if available, could be used daily, and should be a great help in the prevention of respiratory complications in poliomyelitis.

The following two case reports illustrate the use of trypsin instilled endobronchially in cases of chronic atelectasis.

CASE REPORTS

CASE 1—B S, a 31 year old white woman, was admitted to Colorado General Hospital on Nov 5, 1950, with acute bulbar spinal poliomyelitis. Following admission she was placed in a respirator, and during the next five month period her condition improved sufficiently so that she could be gradually weaned from auxiliary respiratory aid. Examination on discharge from the hospital revealed a severe paralysis of all four extremities, but most marked in the arms. There was some weakness of the abdominal and back muscles, and there was slight weakness of the left diaphragm, the right diaphragm was paralyzed, and there was severe weakness of all intercostal muscles. She weighed at this time about 75 pounds. Past history revealed that she had suffered from bronchial asthma for many years, apparently aggravated by her social environment. The patient did not have any attacks of asthma while in the hospital, but described such episodes on the few occasions when she was home on week end leave. On Jan 10, 1952, the patient had a rather severe asthmatic attack. She was hospitalized in her home community and treated with oxygen and antiasthmatic drugs without benefit. Subsequently, she was again transferred to Colorado General Hospital where examination revealed an extremely apprehensive and anxious patient in spite of previous large doses of sedatives. Her respirations were rapid and shallow, and she complained of dyspnea with difficulty in breathing. There was very little movement of the chest cage, and all respiration seemed mainly left abdominal. Breath sounds were diminished over the entire chest, and were absent over the right lower chest. Very few rhonchi or rales were audible. It was felt that the patient was not having an asthmatic attack, but had developed a right lower lobe atelectasis. Fluoroscopy was performed and revealed atelectasis of the middle lobe and portions of the right lower lobe (Fig 1). The patient continued to develop increasing dyspnea and seemed unable to dispose of the accumulating secretions. Consequently, two

Following admission, a tracheostomy was performed. Almost immediately her color improved, but her breathing remained labored. She was placed in an Emerson respirator and spent 8 hours each day on the oscillating bed. This treatment was supplemented with anti-infectives and intermittent positive pressure oxygen. She also received aerosol Isuprel and pleural drainage. However, her condition did not improve on this conservative management. She continued to run a low grade fever and to have difficulty carrying on respiration without aid. On January 16, bronchoscopy was performed (Fig. 2). All segmental orifices were inspected and noted to be normal. There was some accumulated secretion in the left lower bronchus which was aspirated. The bronchoscope was removed to the carina where, under positive pressure oxygen the lungs were inflated. Subsequently there was no change in the clinical or roentgenological findings of the patient. On January 25 a repeat bronchoscopy was performed (Fig. 3). The right main bronchus below the level of the upper lobe bronchus was found to be partially occluded by a large quantity of thick white mucoid secretion. This material was aspirated as fully as possible so that the middle and lower lobe bronchi could be well visualized. Again there was no improvement clinically or roentgenographically. Bronchoscopy was again repeated on January 30. A large thick plug of



One hour later bronchoscopy revealed small amounts of much less viscid secretion in the right main and lower lobe bronchi. This was aspirated so that all orifices were observed unobstructed by any secretions. The patient tolerated the procedure well, and subsequently seemed somewhat improved clinically. Fluoroscopy revealed apparent clearing of the right middle lobe without change in the appearance of the right lower lobe. The same procedure was carried out again on Feb 7. On this occasion a small amount of thick mucoid secretion was observed in the right upper lobe orifice and right main stem bronchus just below the middle lobe orifice. None of the lobar bronchi appeared to be occluded, however. One hour following the introduction of 125 mg of Tryptar a small amount of frothy mucoid secretion was seen in the right lower lobe bronchus. This was aspirated well, after which a good blast of air was obtained from beyond this area. Clinically, the



Fig 2



Fig 3

Fig 2—Roentgenogram Jan 16 1952 taken following bronchoscopy reveals a persistent atelectasis of the right lower lobe.

Fig 3—Roentgenogram Jan 25 1952 taken following second bronchoscopy reveals no improvement in atelectatic process.

patient showed no change in physical signs over the previous procedure. On February 11 the patient was bronchoscoped once more and again a large amount of thick creamy yellowish secretion was found in the right bronchus. This was again aspirated and on this occasion 250 mg of Tryptar in 5 cc of Sorensen's Phosphate Buffer solution were injected slowly into the right bronchus. One hour later a considerable quantity of thin watery mucoid secretion was removed. This appeared to be coming from the right lower lobe. The opinion was expressed that the patient's bronchial tree had shown considerable improvement during the recent courses of therapy with Tryptar. Roentgenographic examination of the

on February 15 (Fig. 4) showed no evidence whatsoever of atelectasis, and clinically the patient was much improved. Fluoroscopy on February 22 revealed a paralyzed right diaphragm with good movement of the left diaphragm. The costophrenic sinus on the right was somewhat obscured but the lung was clear. It was felt that she had some chronic pleuritic thickening. There was paradoxical movement of the right diaphragm during breathing and coughing. This was more marked in the erect than in the supine position. The patient was bronchoscoped again on March 7 at which time the right bronchus appeared normal. Roentgenographic examination at this time again showed no evidence of atelectasis (Fig. 5). The patient was then discharged.



FIG. 4



FIG. 5

hensive, and cyanotic. He showed definite mental symptoms with facial grimacing and profuse salivation. These were attributed to cerebral hypoxia. Since it was impossible to remove the patient from the respirator even momentarily, the clinical signs of atelectasis were difficult to evaluate. It was felt, however, that this was the basic cause of the patient's difficulty. Deep tracheal suction was apparently inadequate. Therefore, bronchoscopy was performed and both main stem bronchi were cleared of a large amount of mucopurulent sputum with resultant improvement in the patient's aeration and color. Three days later this procedure was repeated and again revealed a large amount of mucous material particularly on the left side. Removal of these mucus plugs resulted again in marked clinical improvement.



Fig 6—Roentgenogram taken Nov. 26, 1951, following the fourth bronchoscopy reveals persistent atelectasis of the left lower lobe.

The patient showed little evidence of recovery from his paralysis during the next two month period. He was able to be out of the Emerson respirator for no longer than fifteen minutes. Examination on these occasions revealed dullness over both lower lung fields. On Nov. 16, 1951, the patient developed sudden difficulty in breathing and a rapid, thready pulse. Roentgenographic examination revealed haziness of the entire left lung field with some mediastinal shift to the left. Bronchoscopic aspiration was again performed with subsequent improvement in the patient's general clinical condition. Three days later the patient was fluoroscoped and was observed to have hyperventilation of the right lung. There was some scoliosis with a concavity to the left. Auscultation revealed dullness over the left lower lung field. It was felt that the atelectasis contributed in large extent to the inability of the patient to remain out of a respirator. Repeated bronchoscopy on November 21 allowed aspiration of two small mucus plugs from the left lower bronchus, but the patient showed no clinical improvement (Fig 6). A chest roentgenogram, taken December 27, revealed atelectasis of the left lower lobe still present, with a marked shift of the mediastinum and heart to the left. The right lung now showed definite signs

of apical emphysema. The patient showed some slight improvement during the first month. He was able to be out of the respirator for twenty-eight to thirty minutes at a time. A chest roentgenogram of the chest was obtained on Jan. 25, 1952, and now showed virtually complete atelectasis of the entire left lung (Fig. 7). Examination revealed normal breath sounds corresponding to this area. It was apparent that there was a clinically silent extension of the atelectatic process during the time since the patient one month previously. The patient had not complained of any increased cough, nor did he show any elevation of temperature or other signs associated with an infection. On January 30 the patient was bronchoscoped once more. Although segmental atelectasis had been present for at least five months and the upper lobe could not be inflated for an indeterminate period of time, we felt that it would be worth trying



Fig. 7. Roentgenogram taken Jan. 25, 1952, showing atelectasis of the entire left lung. The patient was unable to breathe without the respirator at this time.

dyspneic, but he did complain of slight pain in the left chest and some burning in the trachea. He was immediately given 50 mg of Benadryl intravenously, and within two hours all symptoms had disappeared. Roentgenologic examination of the chest now showed that the entire upper lobe had cleared but that the atelectasis of the lower lobe remained. Subsequently, roentgenograms on January 31 (Fig 8), February 2, and February 4 did not reveal any further clearing of this area of atelectasis at the left base. However, the patient was able to increase his period out of the respirator to as much as forty five minutes, and his general condition showed marked improvement. One month later a roentgenogram revealed condensation of the atelectatic area of the left base, so that now there existed only a small triangular shadow behind the heart (Fig 9). The impression was that there had been a marked improvement over the previous roentgenograms. On March 10 it was decided to bronchoscope the patient again and on this occasion to inject 250 mg of Trypsin. Following this procedure, a considerable amount of thin, watery mucous secretion was removed from the left lower lobe bronchus (Fig 10). During the following week the patient was able to increase the period of time out of a respirator to as much as one and one-half hours and in two weeks to as much as three hours. Roentgenograms have shown essentially complete resolution of the remaining atelectatic area (Fig 11).



Fig 8—Roentgenogram taken Jan 31 1952 following initial trypsin therapy. Illustrates clearing of left upper lobe atelectasis with no improvement in right lower lobe collapse.

DISCUSSION

It would seem logical to assume that chronic atelectasis should be attacked more vigorously from a conservative standpoint before the stage is reached which will require radical surgical measures. The use of the physiologic enzyme, trypsin appears to offer this possibility. Trypsin has been shown to have a mucolytic action *in vitro* and might reasonably be expected to aid in the removal of inspissated bronchial secretions which have resulted



F 2—Posteroanterior taken March 1, 1952. Illustrates the improvement but partial atelectasis of the left lower lobe persists.



F 3—Posteroanterior taken March 1, 1952. Illustrates the improvement but partial atelectasis of the left lower lobe persists.

in atelectasis. Since it would not be expected to have any effect on the bronchial mucosa, local damage to the respiratory tree is not to be anticipated. However, in the face of acute inflammation of the bronchial tree, some local tissue damage could occur. We have shown that in tissue culture the fibrin matrix holding young fibroblasts together is dissolved and the cells are killed by trypsin in large dosage. In the face of an acute inflammatory process trypsin could, therefore, result in local edema and superficial necrosis. It would perhaps be best not to use the drug in cases of acute tracheobronchitis. Experience has also taught us that it is contraindicated in tuberculosis if a bronchopleural fistula is present or if a fistula is opened as a result of trypsin administration.

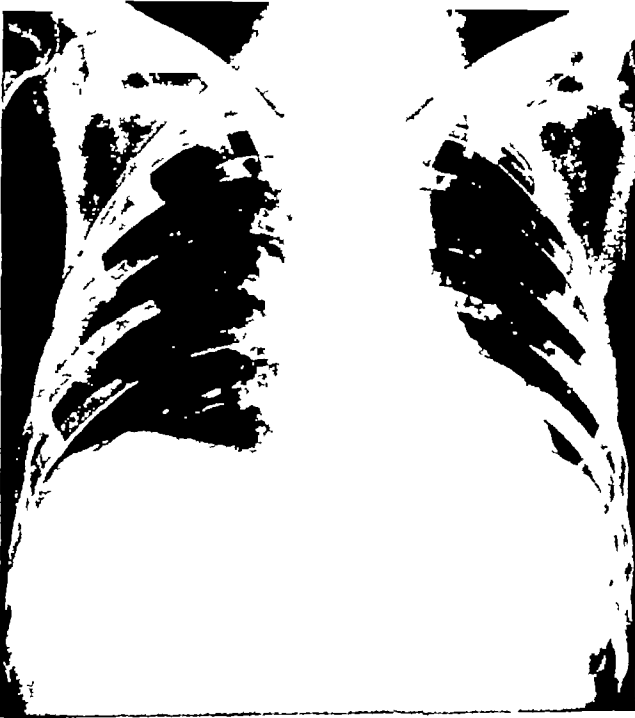


Fig. 11—Roentgenogram April 29, 1952, reveals essentially complete resolution of remaining atelectatic area.

Another source of potential complication might be related to the rapid absorption of trypsin into the blood in large concentrations from the pulmonary parenchyma. It is our feeling that there is no danger in this regard. We have injected trypsin into the heart chambers of animals (rats) in concentrations equivalent to and far above that used clinically, without any evidence of damage or of anaphylactic or histaminic response. Antitrypsinogenase is present in the circulating blood and it is to be assumed that the activity of trypsin is destroyed almost immediately.

We assumed, therefore, and have shown, that the procedure of endobronchial administration of trypsin is an innocuous one physiologically, and that a worthy or more thorough investigation in carefully selected cases of chronic atelectasis due to bronchial obstruction from inspissated mucoid material. We feel that the problem of atelectasis in bulbar poliomyelitis offers the ideal situation in which this type of therapy might be beneficial.

SUMMARY

- 1 The physiopathology of atelectasis is reviewed
- 2 The therapy of atelectasis is considered particularly in the patient with respiratory poliomyelitis
- 3 The use of trypsin by endobronchial installation is described as it has been used in loosening the viscid secretions of patients with atelectasis
- 4 Two case reports are presented illustrating the successful use of this material in relieving chronic atelectasis due to bronchial and bronchiolar obstruction in patients with bulbar poliomyelitis
- 5 It is urged that further experience be accumulated on the effectiveness of endobronchial trypsin in relieving persistent atelectasis in carefully selected patients

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CONCURRENT LYMPHOMA OF THE LUNG AND STOMACH

FOLLOW-UP ON A PREVIOUSLY REPORTED CASE

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The case described herein has previously been reported in this journal as primary lymphoma of the lung.¹ Since that publication what was originally thought to be an associated benign gastric ulcer in an otherwise normal stomach proved to be a recurrent ulcer forming in a stomach that this patient had two lymphomatous ulcers. It would appear therefore that this patient had two lymphomas at the same time. This has been definitely established by operation both in the stomach and the lung and both seem to be following a benign course. This is not one or single primary lymphoma of the lung. It may be a double primary tumor such as is seen at times in epithelial neoplasia or one may be distinct from the other. It is for this reason and the unusual general interest of this case that we make the following report. This case also represents to our knowledge the first published account of a lymphoma of the stomach removed by gastrosopic biopsy and proved by operation.

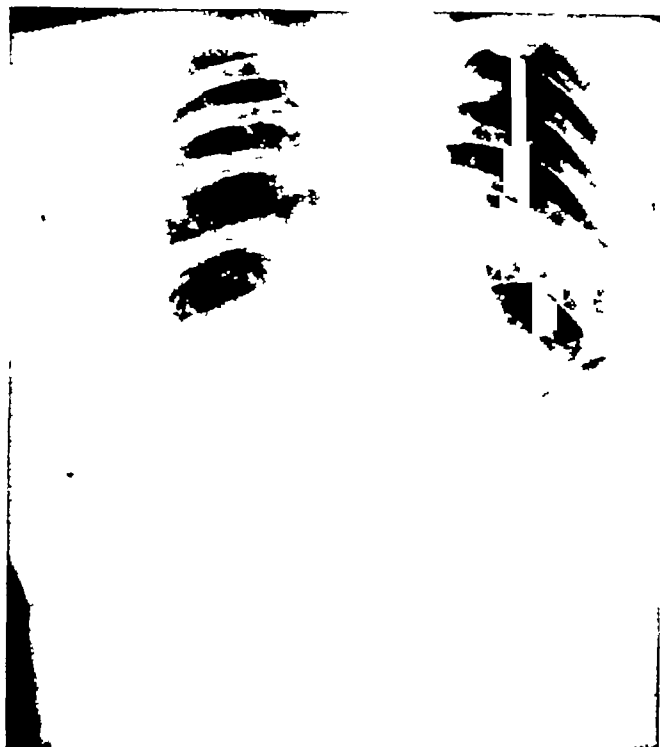


Fig 1—Chest roentgenogram showing right lower-lobe tumor



Fig 2—Gastroduodenal roentg nogram showing ulcer defect of posterior wall



Fig. 2—Biopsy of ulcer showing a few typical gastric glands and massive cellular infiltration first interpreted as probably inflammatory later thought to be a fairly typical carcinoma.



Fig. 3—High magnification of cellular infiltration.

typical of that found in the base of any ulcer (Fig 3) The cells showed no mitotic activity, and it was thought that this tissue represented benign gastric mucosa with considerable cellular infiltration and reaction, and a degenerating ulcer membrane

It was believed by the surgeon that the lung tumor was probably metastatic from a malignant process in the stomach It was decided that the lung be explored first A thoracotomy revealed a firm mass palpable in the lower lobe of the right lung There were no adhesions present, but the tumor was firmly encapsulated There were no other tumors found A right lower lobectomy was performed Microscopic examination of the



Fig 5—Microscopic section of stomach after subtotal gastrectomy showing change identical to that of the lung tumor—lymphoma.

tumor showed that it consisted almost completely of round cells without stroma (Fig 4) It was believed that this was probably a benign lymphoma, and the ulcer of the stomach was benign and not connected with the lung lesion or related to it

The patient made an uneventful recovery Her gastrointestinal symptoms subsided During her convalescence a gastroscopic examination was done and another stomach roentgenogram made Both showed remarkable and complete healing of the gastric ulcer Repeated roentgenograms throughout 1950 and 1951 showed no recurrence of the ulcer, and the chest remained clear

PRESENT ADMISSION

The patient was again seen here in December, 1951, having had some epigastric pains at irregular intervals for several weeks without vomiting, but with the passage of dark stools for several days. Examination showed a pale, haggard appearing patient with mild epigastric tenderness. Otherwise the findings were essentially normal. Her hemoglobin was 10 Gm per hundred cubic centimeters and dropped to 7.5 in the next 6 days. Stomach roentgenogram again showed a thickened irregularity of the greater curve not truly rigid and without apparent crater. At gastroscopy, an ulcer about 1 cm or a 25 cent piece was seen, it was covered with mucus and had clean cut edges. A guaiacum stain of these secretions was negative for tumor cells, and a gastroscopic biopsy showed round cell infiltration and marked inflammatory reaction. A subtotal gastrectomy was performed. Gallstones were found at exploration. The surgeon felt it was unwise to remove them at this time. The stomach was markedly thickened and there were many hemorrhagic areas. No frank ulcerations were seen. A section microscopic examination showed a stomach section to have been replaced entirely by round cells, having the exact appearance of the lymphoma removed from the lung three years previously (Fig. 5). Permanent section examination confirmed this. The patient had an uneventful postoperative recovery and has had no distress up to date.

DISCUSSION

Many questions arise that cannot be answered definitely. Is this a malignant or benign condition? The tumor does not appear malignant either gross or microscopic examination and the patient's clinical course so far is benign. Marshall and Brown¹ have reported many patients who have survived 10 years with this disease. They find that the prognosis is generally better with lymphoma of the stomach than with carcinoma of the stomach. Is this primary lymphoma of the stomach or lung? Did they develop simultaneously? Are they merely part of a generalized lymphomatous condition? One thing is definite. Lymphoma of the stomach was diagnosable by gastroscopic biopsy three years ago. Review of the gastroscopic biopsy specimens removed at that time showed a round cell infiltration which was mistakenly called carcinoma. In light of three years of additional experience with gastroscopic biopsy we believe that this tissue could definitely be diagnosed as lymphoma.

SUMMARY AND CONCLUSIONS

A case of lymphoma of the stomach and lung has been presented. The case as previously reported is one of primary lymphoma of the lung. It is apparent that it was not a single primary tumor. In retrospect we believe that a correct diagnosis could have been reached previously by proper interpretation of the gastroscopic biopsy.

REFERENCES

Announcement

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The 33rd annual meeting of the American Association for Thoracic Surgery will be held March 27, 28, and 30, 1953, in San Francisco, Calif. Headquarters will be at the Hotel Fairmont. Suitable accommodations also have been reserved at near by hotels, the Mark Hopkins, the Huntington and the Sir Francis Drake.

Requests for Hotel Reservations

These should be mailed to Dr. H. Brodie Stephens, Chairman of Hotel Committee, American Association for Thoracic Surgery, Room 200, 61 Grove St., San Francisco 2, Calif. The following information must be included: (1) two choices of hotels, (2) type of accommodation desired—single, double, twin, room or suite, (3) name of other room occupants, if any, whether wife or another doctor, (4) date and approximate hour of arrival, (5) date of departure, (6) return address.

Abstracts for Papers

Abstracts for the presentation of papers at the 1953 meeting must be received on or before Dec. 1, 1952, otherwise they will not be considered by the Program Committee. *Five, repeat, five* copies of each abstract should be sent to the Secretary of the Association, Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif. They should not be sent to the Editor.

The abstracts should contain from 200 to 250 words, and should accurately reflect the contents of the completed paper.

Applications for Membership

Applications for Associate Membership in the American Association for Thoracic Surgery must be received by the Membership Committee not later than December 1, 1952, to be considered for action at the meeting in San Francisco in March, 1953. Applications received on a date beyond that deadline must be deferred for consideration until 1954.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the sponsoring statement on the application form, the sponsors must submit a separate letter concerning the applicant direct to the Chairman of the Membership Committee. These regulations are at the direction of the Council and were made effective at the meeting in Dallas in 1952.

Completed application forms should be sent to the Chairman of the Membership Committee, Edward M. Kent, M.D., 1406 Clark Building, Pittsburgh 22, Pa.

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